The New England
Journal of Medicine

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Volume 231 SEPTEMBER 14, 1944 Number 11

PSYCHIATRIC CASUALTIES OF WAR AND THEIR TREATMENT*

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IT IS no exaggeration to say that in the present war the most important medical problem is that of the treatment and prevention of psychiatric casualties. Certainly those who are familiar with the experience of World War I will not be astonished by such a statement. To date the United States Government has spent well over $1,000,000,000 for the compensation and hospitalization of the nervous and mental casualties of World War I, and even now about 67,000 veterans of that war are receiving compensation for psychiatric disabilities that have been adjudicated as due to service. As a result of the legislative provision that veterans of 1917 and 1918 are eligible for care whether or not their disability is due to service, the psychiatric hospital peak load for World War I has not yet been reached. Whether the rate of incidence of neurologic and psychiatric disorders is greater in this war than it was twenty-five years ago is at the moment uncertain. It may, however, be safely said that on account of the vastly greater size of the armed forces, the number of cases developing must be large. Certain figures published in the January 14, 1944, issue of the *Congressional Record* indicate that out of a total of 3,437,000 registrants rejected from the draft, those rejected for mental disorders constituted 14.5 per cent, those for neurologic disorders 5.4 per cent and those on the ground of mental deficiency 3.3 per cent.

From the experience of World War I it was the hope and expectation that a careful selection of registrants at the induction level would bring about a lowering of the discharge rate for nervous and mental disorder. Unfortunately, that expectation has not been fulfilled. According to the same issue of the *Congressional Record*, the Selective Service System reported that up to January 1, 1944, of the discharges from the Army, 44.6 per cent have been for neurologic and psychiatric conditions. Of these, psychoneurosis led, with 32 per cent of the discharges. Indeed, the next most frequent cause, schizophrenia — or, as it is sometimes still referred to, dementia praecox — constituted only 4.1 per cent of the total. Epilepsy and neurologic disorders accounted for 2.2 per cent each, and other psychiatric conditions were even less numerous.

The person who leaves civilian life to enter the Army finds himself faced by a number of psychologic hazards.[1] One of the greatest of these is the loss of individuality, but there are others. The strict regimentation, the loss of initiative and privacy, the sudden deprivation of dependency on home and family, the sense of loneliness and homesickness, are all factors that are prominent in studies of the psychiatric casualties. The alteration in attitudes toward human life calls for a radical readjustment and reassessment of values. To all these hazards are added, particularly under combat conditions, various physiologic insults caused by mud, vermin, various diseases and loss of sleep and, above all, by sustained fear. Under these added stresses it is perhaps strange that more men do not develop a condition in which their psychologic equilibrium can no longer be maintained. On the compensating side it should be pointed out that there are certain constructive phases of military life. The soldier has excitement and change, and he is freed of many of his civilian problems and of the various frustrations of civilian life. If he feels confidence in his leaders and in the cohesiveness of his organization, his own feelings of security and power are increased. It is this mechanism that operates so powerfully under the name of "morale"— perhaps the most important single factor in the rate of incidence of neurosis. There is an identification with the interests of the country in duty and service, and new authority is gained, together with greater maturity. Some neurotic persons, particularly those of an obsessive compulsive type, may fit in particularly well to this picture. Certainly one can say that even though the balance in war is always on the debit side, there are constructive factors, and obviously the majority of men make a

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 23, 1944.

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working adjustment to the stresses that they meet in military life, just as they do to those encountered in civilian life.

Although the psychologic factors involved in the military situation are of a special nature, the reactions to them follow general patterns that are familiar to psychiatrists in civilian practice. Human nature, in short, does not change either in its aims or in its manners of defense. As a general thing it may be said that the psychiatric reactions of war tend to be somewhat more superficial, to run a shorter course and to be more readily amenable to treatment, particularly removal from the specific situation, than is the case with similar types of reaction occurring among civilians.

The psychotic reactions — that is, those reactions that in civilian life would ordinarily call for commitment to a mental hospital — may be dismissed in a comparatively short space. Probably the most frequent psychotic reaction is what is generally diagnosed as acute catatonic excitement or, as Kempf would term it, acute homosexual panic. This condition arises not infrequently in the early days of camp life and is characterized by a sudden onset with marked overactivity, fearfulness, confusion and active auditory hallucinations in which the voices accuse the patient of homosexual practices or inclinations. There may be assaultiveness, and there is frequently refusal of food, which, in connection with the overactivity, may bring about an early dehydration, exhaustion and occasionally even death. It seems likely that many of these reactions are precipitated in latent homosexuals who suddenly find themselves in the midst of large numbers of others of their own sex with little or no privacy. This type of reaction is far from unknown in civilian life, and the recovery rate in state hospitals is not insubstantial. The response to hospital care, particularly with prolonged sedation and interviews with the patient under the influence of Sodium Amytal, is substantially higher and the course, although stormy, is usually brief.[2] Among the other psychoses are found more rarely depressions, and somewhat more frequently mild manic episodes. These, too, are usually readily responsive to treatment.

Among the psychiatric casualties of war, the psychoses pale into insignificance as compared with the psychoneuroses. In connection with the neuroses an observation may be made about what may be termed "the styles." Just as the *grande hystérie* of Charcot had become practically obsolete by the time of World War I, so it is now found that the trend in the neuroses — at least in the military sphere, and probably in the civilian as well — is away from the conversion hysterias, which were frequent in 1918, in favor of the anxiety states. Anxiety neurosis clearly leads the list among the neuroses. In this condition there is a prevailing mood of worry, with acute attacks of free-floating or unfixated anxiety, accompanied by the autonomic symptoms of flushing, sweating, tremors, palpitation and dyspnea, the startle reaction and disturbances of sleep, often accompanied by terrifying dreams. This condition, although not confined to the combat areas, is generally attributed to some catastrophic event, such as an explosion near by, a torpedoing or the death by violence of a comrade. It should always be borne in mind that any person may, if the stress is sufficiently prolonged and sufficiently severe, develop a state of this sort. No one is immune to the neuroses. Often the fundamental conflicts are found not to be the fear of immediate dissolution, but rather to concern domestic situations or other civilian problems, some of which have evidently been in existence for a long period. In general it may be said, just as was found in the follow-up of a group of psychoneurotic patients after World War I, that the more clearly exogenous are the precipitating factors, the better are the prospects. It was found, for example, in 1918 that those victims of neurosis who went back to duty in the largest proportion were those who had been exposed to the concussion of high explosives; their conditions were diagnosed, for example, as concussion syndrome, traumatic syndrome and exhaustion neurosis. A far smaller proportion of those with hysteria were returned to duty than was the case with those with relatively mild and superficial anxiety states that developed following a clear-cut precipitating episode.

Conversion hysteria, which was one of the ordinary conditions in 1917–1918, is relatively less frequent today, although not by any means unknown. A number of conditions, somewhat allied, are grouped under the current heading of "psychosomatic disorders." Effort syndrome, in which one found attacks of dyspnea, sweating, tremulousness and precordial pain, was described by Da Costa soon after the Civil War under the title of "soldier's heart," and was recognized even then as presumably functional in origin. It was frequent in World War I, but is probably somewhat less so in the present conflict. It is generally recognized that a considerable group of gastrointestinal disorders are likewise essentially emotional in origin. This group is not larger entirely on account of the increased diagnostic acumen of the internists, although with the increasing stress now being laid on the importance of psychosomatic medicine this is a factor that cannot be disregarded.

The list of disorders that have been discussed is far from exhaustive, but it represents the principal types of psychiatric casualty likely to be encountered in the military experience. The degree of severity of the symptoms varies, as do some of the specific situations in which certain of the symptoms develop. One thinks, for example, of some of the special problems of aviation medicine, of tank warfare and of fighting in the jungles. The mechanisms, as may

properly be repeated, are fundamentally the same, although the precipitating conditions are different. It is extremely doubtful whether any useful purpose is served by coining new names for these conditions. One hears much nowadays of combat fatigue, fatigue syndrome, flying stress, operational stress and so on, just as one heard of shell shock in World War I.[3] These attempts to establish a new terminology apparently stem from the idea that there is something inherently disgraceful or improper about the development of a psychoneurosis. If the same amount of energy were put into educating the military and the public concerning the true nature of neurosis as is now devoted to the coining of new names, we should probably be making far more progress!

In a recent issue of a widely read magazine that has never been particularly noted for the accuracy of its medical articles, the readers were informed that there was no curative treatment of psychiatric casualties in World War I, that very few of the men recovered, and that as a result the psychiatric hospitals of the Veterans Administration are now crowded with this "débris of war," whereas a curative treatment has now been developed that is redeeming casualties for combat or useful noncombat duties. The remarks concerning World War I are an interesting example of the manner in which Americans characteristically forget the past. Documentary evidence is readily available that a great deal was done in 1917–1918 in the line of the early treatment of neuroses near the front lines, and it is likewise clear that a very substantial number of men were sent back within ten days or less to front-line duty.[4] During the Meuse-Argonne drive, for example, 75 per cent of the men treated for war neuroses at the field hospital were returned to duty in an average of four days, and only about 4 per cent of recurrences were found. Rest, occupation and psychotherapy, the very things that are being stressed today, one finds emphasized then. This is not to say, however, that no progress has been made in the line of therapy. Much, indeed, of a constructive nature has taken place.

Much has been learned since World War I of the psychologic mechanisms of these casualties, and there have been learned, too, several new approaches to the patient that enable psychotherapy to be more effective. Among the newer methods perhaps the most significant is what has been variously termed "narcoanalysis" and "narcosynthesis" or "Amytal interviews." The first term was invented by Horsley[5, 6] in 1936, and the second has been utilized more recently by Grinker[7] in describing his significant work in the North African campaign. The treatment consists in the intravenous administration of Sodium Amytal or Pentothal Sodium in a dose sufficient to bring about a moderate degree of narcosis (5 to 7½ gr.). This treatment is a valuable aid in releasing inhibitions, overcoming the resistance of the patient and enhancing his suggestibility. It may be utilized in the treatment of the neuroses and even of the psychoses, particularly when the patient is inaccessible. It provides an access to the deeper layers of consciousness, and not infrequently a patient talks freely who without the drug would be completely mute. In the stage of suggestibility brought about immediately after the administration of the drug, hysterical symptoms may be caused to disappear, and it can then be demonstrated to the patient that his disability is not a permanent one. Narcoanalysis is a valuable procedure that is being widely used with great success. It requires no apparatus except the hypodermic syringe, can be utilized at points near the front lines and has been used in a large number of cases in restoring soldiers to active duty. It should always be remembered, however, that the drug constitutes merely a useful adjuvant to psychotherapy. The important feature of this method of treatment is the increased accessibility of the patient to the ministrations of the therapist; to this extent, it really should hardly be classified among the drug therapies.

Continuous narcosis treatment calls for hospital facilities and a good deal of nursing attention. It has not been so promising as was hoped when it was first introduced, and it is likely that the simpler method of narcoanalysis provided all the benefits, except that in cases of profound exhaustion the continued narcosis provides a long period of much needed rest.

A few words should be said about the various types of what Strecker[8] has aptly termed "the drastic therapies." Insulin-shock therapy is not especially applicable in these cases, although Sargant[9] has used what he terms "sub-shock insulin therapy" and has found it useful as a general tonic in cases of war neuroses, and also as a sedative therapy in excited states. The Metrazol treatment of Meduna has been found to have certain disadvantages and has been almost entirely replaced by the electric-shock treatment of Cerletti. The latter has been particularly useful in the treatment of the depressions, and is therefore applicable largely in general or station hospitals rather than nearer the front lines. In the depressions there is always danger of suicide, hence any treatment that shortens the course of the disorder not only benefits the patient but results in a saving of available man power among the hospital personnel. The electric-shock method is not without risk, however, and should not be employed without the precaution of a thorough physical examination, in addition to x-ray examination of the chest and spine, and preferably (at least in patients past thirty) an electrocardiogram. Obviously physiologic changes take place when the electric current is passed through the brain of the subject. I find it hard, however, to agree with Myerson[10] who has stated

categorically that the value of shock treatment does not appear to have a psychologic foundation. He proceeds to say, rather, "Physiologic alterations of an unknown type take place and this is the basis of the recovery." To many of us one of the values of the shock treatments in general and of electric-shock in particular has seemed to be that it placed the patient in an especially dependent situation with relation to the physician, and that in turn it directed the physician's attention to the needs of the patient, thus bringing about a closer rapport. Indeed, electric-shock or other shock therapy, without psychotherapy, — that is, the impact of the personality of the physician on the patient,— is likely to fail of its full effect. There is no royal road, even an electrical one, to the cure of mental disorders, but there is no doubt that electric-shock therapy has been of great value, particularly in the treatment of the depressions. It has likewise been found useful in acute maniacal conditions.

Years ago Pratt[11] demonstrated effectively what could be done in the line of group therapy. The work that he started is being confirmed and developed in many different ways today. Psychotherapy is a time-consuming process. The number of persons who need it is large, and the floating supply of psychiatrists is small. Two things must be done if adequate treatment is to be given to the patients being considered. One is to develop in nonpsychiatrists an appreciation of the importance of emotional conflict and the need and the value of psychotherapy. The other is to arrange for the treatment of groups of patients, thus multiplying for practical purposes the amount of time that the therapist may employ per patient. Group discussions are being used at various centers with substantial success, and at a few points a specialized form, the psychodrama, has proved successful.

Among other forms of therapy one certainly must not fail to mention occupational therapy, a form that was utilized overseas during World War I, and which is again being developed by the Army. It seems probable that its value will eventually be fully recognized again, as it was in 1918. Various physiotherapeutic approaches, some of which have already been mentioned, are of value, partly on account of the physiologic changes brought about by them, and partly on account of the psychologic factors involved.

Time does not permit a more detailed consideration of a long and complicated subject. Enough has been said, however, to indicate that although the incidence of psychiatric casualties is substantial, the prognosis, both for the psychoses and for the neurotic manifestations, is better than one would expect to find with similar symptoms in civilian life.

What of the future? One of the greatest defects of public education at the moment is shown in the lack of the proper attitude toward the psychiatric dischargee and rejectee. The attitude of the family, the attitude of the employer and the attitude of society as a whole leave much to be desired. Too many persons fear the man who is known to have suffered a psychiatric disability in service. They fear that he is a "raving maniac," that he is unsafe to have about, that he is so changed a person that he should be looked on as a pariah. There are employers who refuse to re-employ a man known to have been discharged for a psychiatric cause. Such a situation is tragic and must be rectified if the improvement of these men is to be maintained. If the dischargee comes home to find himself rejected, he loses confidence and may readily suffer a relapse. One may question also what the effect of the generous compensation laws enacted by Congress is to be. It may well be that cases will be kept active because recovery is to be penalized by loss of a fixed income. In this regard war casualties of a psychiatric nature are likely to be similar to unsettled industrial-compensation cases. If lump-sum settlements could be arranged, it is likely that greater inducement would result for the recovery of the patient. Finally and most important, if these men on returning can be made to feel that they are respected, that they are wanted, that their place in the community is secure and that work is available for them, we may safely dismiss any fears that we are to be overwhelmed by a flood of permanent psychiatric casualties of war.

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POTASSIUM BICARBONATE: AN ADJUNCT TO CHEMOTHERAPY IN PNEUMONIA COMPLICATING CARDIAC DECOMPENSATION*

A Preliminary Report

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A COMPLICATION frequently encountered in the administration of sulfonamides is kidney damage. If adequate urinary output is maintained and the urine is kept alkaline, the incidence of crystalluria, hematuria and frank renal damage is decreased.[1, 2] These measures are particularly important in patients with marked cardiac insufficiency, who tend to retain ingested water and to excrete small amounts of urine.

Sodium bicarbonate is most frequently used for alkalinization. Modern therapeutics, however, stresses the primary significance of sodium retention in the development of cardiac edema and congestive heart failure.[3-7] In cardiac decompensation, a regime promoting a negative sodium balance is considered advisable; it is not aided by the administration of sodium bicarbonate.

In the medical wards of this hospital are many patients in whom pneumonia accompanies and often precipitates cardiac decompensation. Rational therapy requires that both be treated simultaneously. This involves the administration of sulfonamides, the maintenance of adequate urinary output, urinary alkalinization, restriction of sodium intake and promotion of diuresis, as well as the employment of other recognized measures.

Because the administration of sodium bicarbonate was considered to be incompatible with these objectives, other alkalinizing agents were investigated. Many were tried and found to be unsatisfactory because of their tendency to cause gastrointestinal upsets when used in sufficiently large amounts. Potassium bicarbonate was finally chosen for clinical trial, since it was well tolerated in required doses.

METHOD OF STUDY

A group of patients with severe cardiac impairment and pneumonia treated with sulfadiazine and potassium bicarbonate were compared with a similar group treated with sulfadiazine and sodium bicarbonate. All the cases studied were admitted to the medical wards of the Greenpoint Hospital between December 1, 1943, and March 1, 1944. The criteria for inclusion in the study were survival for at least seventy-two hours after admission, cardiac decompensation of sufficient severity to be classified Grade III or IV according to the specifications of the American Heart Association,[8] demonstration of a lobar or bronchopneumonic process by clinical and radiologic findings and the absence of findings suggesting severe renal impairment or adrenal insufficiency. The last criterion was adopted as a precaution against potassium toxicity. Patients treated by us were given potassium bicarbonate; those treated by the other members of the house staff were given sodium bicarbonate.

All patients were given an initial dose of 4 gm. of sulfadiazine, followed by 1 gm. every four hours. In addition, they received an initial dose of 8 gm. of sodium or potassium bicarbonate, followed by 2 gm. of the chosen agent every four hours. To both groups fluids were given freely, and the patients were encouraged to drink at least 2500 cc. per day. Daily fluid intakes and outputs were measured, and the urinary pH was determined daily from a 6 a.m. specimen by the nitrazine method. Because of the difficulty of obtaining serum potassium levels, these could not be studied. In addition, many patients were critically ill and weight variations were unobtainable. Further routine orders included complete bed rest, a soft, low-salt, 1200-calorie diet, digitalization and other symptomatic measures as indicated, except diuretics.

RESULTS

Sixteen patients were studied over a total of sixty-five hospital days. Nine patients were treated with potassium bicarbonate (Table 1) and 7 with sodium bicarbonate (Table 2). The intakes, outputs and differences between the two for the two groups are compared in Table 3.

TABLE 1. *Average Daily Fluid Intakes, Outputs and Intake-Output Differences for Patients Treated with Sulfadiazine and Potassium Bicarbonate.*

CASE NO	SEX	AGE	PERIOD TREATED	FLUID INTAKE	FLUID OUTPUT	INTAKE-OUTPUT DIFFERENCE
		yr.	*days*	*cc.*	*cc.*	*cc.*
1	M	66	9	1390	1010	380
2	M	22	3	1620	1010	610
3*	M	69	4	1310	1100	210
4	F	82	5	2090	990	1100
5	F	49	3	3400	1340	2060
6	F	54	7	2970	2820	150
7	F	62	5	2520	910	1610
8	F	46	4	2460	1430	1230
9	F	56	3	1610	1380	230
Averages		56.2	4.2	2152	1332	820

*Patient died.

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Maintenance of alkalinity. The 6-a.m. urine was chosen as the likeliest to be acid, and was taken immediately after awakening, thus avoiding the so-called "morning alkaline tide" of the urine, which is induced by the respiratory readjustments consequent on awakening. In only two of sixty-five samples studied was an acid urine found, and in a great majority a reaction of pH 7.4 or higher was maintained. The dosage of bicarbonate was not increased for patients not showing a urine of pH 7.4, but in further studies it is intended routinely to increase the dosage by 25 per cent to ensure alkalinization.

TABLE 2. *Average Daily Fluid Intakes, Outputs and Intake-Output Differences for Patients Treated with Sulfadiazine and Sodium Bicarbonate.*

Case No.	Sex	Age	Period Treated	Fluid Intake	Fluid Output	Intake-Output Difference
		yr.	*days*	*cc.*	*cc.*	*cc.*
10	M	31	5	1150	720	430
11	M	48	3	2680	1000	1680
12	M	38	3	2030	940	1090
13	M	70	3	1430	260	1170
14	M	76	7	1560	980	580
15*	M	61	2	2430	620	1860
16*	M	81	4	1700	830	870
Averages		60.7	3.9	1861	764	1097

*Patient died.

TABLE 3. *Comparison of Intakes, Outputs and Intake-Output Differences of Patients Treated with Sulfadiazine and Sodium or Potassium Bicarbonate.*

Type of Patient	Fluid Intake	Fluid Output	Intake-Output Difference
	cc.	*cc.*	*cc.*
Potassium-treated patients	2152	1332	820
Sodium-treated patients	1861	764	1097
Differences	291	568	-277
Percentages (K:Na)	116	174	75

Fluid intake. In most cases, it was not possible to achieve the desired daily intake of 2500 cc. The average fluid intake of patients treated with potassium bicarbonate was 2152 cc., and that of those treated with sodium bicarbonate was 1861 cc. The potassium-treated patients thus ingested an average of 291 cc. more than those in the control group.

Fluid output. No patient treated with sodium had an average daily output of over 1000 cc., whereas 7 of the 9 potassium-treated patients exceeded this figure. The average daily output was 1332 cc. in the potassium-treated group, but only 764 cc. in the sodium-treated group. Although, on the average, the former patients consumed 291 cc. more liquid than did the latter, they excreted 568 cc. more daily.

Intake-output differences. Since both groups of patients were maintained under identical conditions of diet, bed rest and environmental temperature, any differences due to amounts of metabolic fluid derived from food and to quantities lost during respiration, from the skin and in the feces were not important and tended to offset each other.[9] Under these conditions the patients showing a marked difference between intake and output were either retaining fluid in the body or excreting body fluid less rapidly than the others. If the body weights of both groups were gradually decreasing, it could be assumed that the observed intake-output difference was due to greater diuresis in one group than in the other.

Since most of the patients recovered from their congestive failure, it seems probable that the observed intake-output difference between the sodium-treated and potassium-treated groups depended on more rapid elimination of retained water by the latter. Unfortunately, daily weight determinations were impractical.

In this series the average difference between intake and output was 820 cc. a day for the potassium-treated patients and 1097 cc. for those treated with sodium. If the difference of 277 cc. a day between the two groups is interpreted as indicating more rapid excretion of excess water by the potassium-treated patients, this means that over the average period of treatment these patients excreted an average of 1250 cc. more retained fluid than did the sodium-treated patients. Only once in the sodium-treated group was the daily urinary output greater than the fluid intake, but outputs greater than intake were observed in one fifth of daily observations in the potassium-treated group.

Discussion

There has been increasing realization that alkalinization of the urine and maintenance of a satisfactory fluid output are essential during the administration of sulfonamides.[1, 2] Approximately 1 per cent of patients receiving sulfadiazine show a significant increase in nonprotein nitrogen blood levels[10]; hematuria and crystalluria are frequent. Sodium bicarbonate given in doses equal to those of sulfadiazine and sulfathiazole reduces the incidence of crystalluria by 60 per cent. The amounts of sulfadiazine and of acetylsulfadiazine dissolved in normal urine at pH 7.5 are twenty times greater than those dissolved at pH 5.0.[2] Despite its theoretical advantage, sulfamerazine is not appreciably less nephrotoxic than sulfadiazine.[11] Alkalinization is usually accomplished by giving 12 to 18 gm. of sodium bicarbonate a day. In most normal subjects the fluid output can be satisfactorily established at over 1000 cc. by an intake of 2500 to 3500 cc. a day.

The low-salt diet given these patients contains about 1.5 gm. of sodium a day. When 12 to 18 gm. of sodium bicarbonate are given daily, another 3.5 to 6.0 gm. of sodium is added. A patient with severe cardiac disease kept on a low-salt diet but

given sulfonamide and sodium bicarbonate receives from 5.0 to 6.5 gm. of sodium each day. This is appreciably larger than the 2-gm. to 4-gm. sodium content of the average American daily diet. Despite general recognition of the importance of sodium restriction in preventing and treating cardiac decompensation, the danger of administering such large doses of sodium bicarbonate to patients with severe heart disease has not been completely discussed by the advocates of alkali therapy.

The physician attempting simultaneous treatment of severe cardiac lesions and pneumonia has had to choose from several difficult alternatives. Sulfadiazine and sodium bicarbonate have been given, achieving alkalinization but risking the impeding of treatment directed toward improving the cardiac status and seriously decreasing the urinary output. Prescription of sulfadiazine without sodium bicarbonate is more considerate of the failing heart, but urinary acidity increases the threat of renal damage. If the fluid intake is limited to the amount required to overcome water retention induced by administered sodium, dangerous minimal fluid outputs result. Under satisfactory conditions patients with severe heart damage tend to have a small urinary output; under stress of infection the output may decline appreciably. This makes alkalinization indispensable when chemotherapy is given to such patients.

The efficacy of potassium bicarbonate in promoting adequate urinary output and rapid elimination of retained water depends on the special differential metabolism of sodium and potassium in cardiac decompensation. The primary factor involved is apparently the tendency of patients with chronic congestive failure to retain sodium.[4] The cause is either endocrine or, more probably, renal; the exact mechanism is not understood, but essentially in all patients there is inability of a functionally damaged heart to meet the metabolic demands of an active person. With retention of sodium, water is held to maintain the constancy of the body's electrolyte pattern and, more importantly, the pattern of the extracellular fluid. Both the plasma volume and the volume of the extracellular, extravascular compartment are increased. Dilution of plasma proteins leads to increased production, with maintenance of normal oncotic pressure. Eventually, a continual increase in plasma volume leads to engorgement of the entire vascular bed, producing increased venous pressure, edema and clinical congestive phenomena. In acute cardiac decompensation other factors lead to increased venous pressure.[3-5] In these patients, both acute and chronic factors were usually clearly implicated.

Pulmonary engorgement, deficient nutrition and the poor resistance of waterlogged, improperly oxygenated tissue lead to increased susceptibility to pneumonic processes. These infections induce secondary factors that further decrease cardiac efficiency. Toxic myocarditis may reduce the already minimal cardiac reserve, and increased respiratory effort and accelerated metabolism of fever increase the load that must be borne. Lung involvement may lead to poor blood oxygenation, with resulting impairment of all oxidative processes, including those of the heart.

During pneumonia there is a decreased chloride output,[12, 13] and with this retention of fixed acid there is a concomitant tendency to retain fixed base. Since sodium is the most important mobile fixed base,[14] this further accentuates the tendency to its retention, and decreases in sodium output result. Moreover, electrolyte concentrations of the extracellular fluid, particularly in lobar pneumonia, are temporarily stabilized at lower values.[15-17] Even if no additional sodium were added to the body stores, additional water could be retained because of decreased sodium concentration per unit volume. It is not surprising that pneumonia so frequently causes striking impairment of the status of patients with injury to the heart.

Unlike sodium salts, which favor water retention, the salts of potassium have a definite diuretic action.[7, 18-20] Both in the normal state and in decompensation, ingested potassium is rapidly and quantitatively excreted, carrying water with it. The excretion of sodium is also somewhat accelerated because the sodium-conserving ability of the kidney is not absolute and because, with elimination of water, a certain amount of sodium must be excreted to defend the electrolyte pattern of the extracellular fluid.[14]

Reports of toxic effects attributable to therapeutic doses of potassium are infrequent, and no signs of intolerance were noted in this series. The oral administration of 40 gm. per day to patients with myasthenia gravis has produced no untoward effect.[20]

The normal serum potassium level is from 3.4 to 4.8 milliequiv. per liter[20]; elevated levels have been reported in shock, asphyxia, hemorrhage, intestinal obstruction and fistula, crush syndrome, pemphigus, severe adrenal insufficiency and uremia.[20-22] Only the latter two had to be considered in this study, and patients in whom either was suspected were excluded. In addition, electrocardiograms were studied, urinary outputs were carefully watched and pulse rates were frequently charted. The patients were observed and questioned about attacks of weakness and gastrointestinal disturbances. Potassium was to be discontinued if electrocardiographic changes, a pulse rate below 70, a urinary output below 600 cc. or an attack of weakness or gastrointestinal intolerance occurred; fortunately, it was never necessary to apply this rule.

In Addison's disease, there is a tendency to lose sodium and to retain potassium[23]; if the salt intake is restricted and potassium is given, an acute exacerbation results.[24] Although patients were excluded

in whom adrenocortical insufficiency was suspected, it is unlikely that our regime could have induced acute adrenal crisis in the decompensated cardiac patients. Such persons to some extent have an electrolyte metabolism opposite to that of the Addisonian; they have ample sodium reserves, their adrenal glands are not impaired, and they tend to sodium retention rather than to sodium depletion.

Toxic effects have been reported in severe renal insufficiency when potassium was given; these consisted of intermittent bradycardia, attacks of weakness and electrocardiographic findings of partial heart block with T-wave inversions.[25] Retention of potassium in uremia is not due merely to the extreme renal damage, for a case has been reported in which uremia was accompanied by potassium depletion.[26] The situation is somewhat analogous to that of renal rickets; in the usual form, there is hyperphosphatemia and hypocalcemia, but rarely hypercalcemic renal rickets is seen.[27] A complex metabolic disturbance occurs in renal insufficiency, but for our purposes it was felt safest to exclude these cases.

If subsequent employment of mercurial diuretics is contemplated, alkalinization is terminated as soon as possible in order not to decrease their efficiency.[28, 29]

Actually, the 12 gm. of potassium bicarbonate given each day represents less potassium than the 10 gm. of potassium chloride frequently used as a daily diuretic dose. Together with the low reported toxicity, adverse effects seem unlikely even without the precautions adopted in this study.

Summary

Potassium salts have long been used as diuretics. Employing conventional dosage, we have attempted to utilize the diuretic effect of potassium and the alkalinizing effect of its bicarbonate to facilitate treatment of patients with severe heart damage who were receiving sulfadiazine for pneumonia. Patients so treated drink somewhat more liquid than do those given sodium bicarbonate, but they excrete almost twice as much urine and eliminate over 250 cc. more of retained fluid each day. By promoting excretion of retained water and sodium, potassium facilitates the re-establishment of cardiac compensation.

At present, we give potassium bicarbonate to all patients whose cardiovascular status is classified as Grade III or IV whenever they receive sulfonamides. Its use is being extended to milder cases as prophylaxis against decompensation. Ten grams is given with the initial dose of sulfonamide and 2.5 gm. every four hours thereafter until twenty-four hours after discontinuance of chemotherapy.

Although apparently of low toxicity, potassium bicarbonate is not used when severe renal impairment or adrenocortical insufficiency is suspected.

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FULMINATING MENINGOCOCCEMIA

Demonstration of Intracellular and Extracellular Meningococci in Direct Smears of the Blood

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IT IS the purpose of this paper to call attention to the unusual finding of meningococci by direct examination of the peripheral blood smear, in a case of fulminating meningococcemia that may also have been a case of the Waterhouse–Friderichsen syndrome.

CASE REPORT

A 27-year-old woman (M. C. V. A-18280) was admitted to the hospital on January 29, 1943. She stated that she was traveling by bus from Atlanta, Georgia, to Elmira, New York, when she began to feel "sore all over," apparently from sleeping in a cramped position. In the course of a few hours there developed a dull headache, stiffness of the neck, slight nausea and finally persistent vomiting. Because of the vomiting the patient left the bus and presented herself at the Emergency Ward of the hospital.

The patient was able to give a clear account of herself, and stated that prior to 8 hours before admission she had been perfectly well. The only significant point in the recent history was the ingestion of ground meat 2 days previously.

Examination showed a well-developed and well-nourished woman lying comfortably in bed. The temperature was 102° F., the pulse 90, the respirations 20, and the blood pressure 100/60. The pupils were equal and reacted to light and accommodation. The ocular fundi showed no papilledema. The pharynx was slightly injected and the patient mentioned a slight feeling of scratchiness. The chest was clear. The heart was normal in size, rate, rhythm and sounds. The neck was supple and no stiffness was noted. The reflexes were equal and active, the Kernig and Brudzinski signs were absent, and the Babinski responses were plantar. The muscles of the entire body were tender on palpation, especially in both lower legs. There was no edema.

The patient appeared to be suffering from influenza, but the diagnoses of trichinosis and meningococcal meningitis were also entertained. Trichinosis was considered because of the ingestion of ground meat of questionable origin, and meningitis because of the prevalence of meningococcal infections.

Examination of the blood revealed a hemoglobin of 76 per cent (Sahli). The white-cell count was 11,900, with 99 per cent neutrophils, most of which showed marked toxic granulation. A lumbar puncture was done in the 4th lumbar interspace and clear fluid under 100 mm. of water pressure was obtained. The cell count was 7 lymphocytes per cubic millimeter. No organisms were seen on microscopic examination of the fluid.

Four hours later a venipuncture was done to obtain a specimen for routine chemical study, and during this procedure several petechiae were noticed on the forearm. No other petechiae were observed, and in view of the normal spinal-fluid findings no significance was attached to this observation.

The following morning the patient was obviously more ill, complaining of severe pains in the muscles and joints and presenting a generalized purpuric rash, with lesions varying in diameter from 0.5 mm. to 2 or 3 cm. These lesions were not raised above the surface of the skin, were nontender and did not fade on pressure. The larger lesions were deep purple, and several lesions of this type were located at the fingertips, simulating closely the appearance of gangrene. Purpuric spots were present over the face, the extremities, the body, the conjunctivas and the oral mucous membrane. A diagnosis of fulminating meningococcemia was at once made. The patient was still mentally alert but was frankly apprehensive, whereas the night before she had regarded herself as only mildly ill.

A blood specimen was taken for culture and examination and at the same time 10 gm. of sodium sulfadiazine was given intravenously. The temperature was 103°F., the pulse 110, and the respirations 24. The white-cell count again revealed 99 per cent neutrophils. There were few adult neutrophils as compared with the number of premature forms, and on stained smears, 7 per cent of the polymorphonuclear cells were found to contain phagocytized gram-negative diplococci (Fig. 1). Extracellular organisms were also observed.

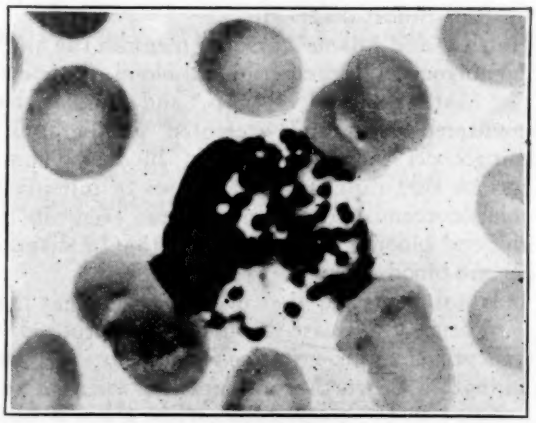

FIGURE 1. *Photomicrograph of a Polymorphonuclear Leukocyte Containing Phagocytized Meningococci (x900).*

At noon the patient was given a 500-cc. transfusion. Antimeningococcus serum was not available. An infusion of 1500 cc. of 5 per cent glucose in physiologic saline solution was given, but the patient became steadily worse. The temperature declined, the pulse rate rose, and the blood pressure fell to 60/40. Four cubic centimeters of cortical extract was given intramuscularly. The purpuric spots previously noted were seen to enlarge and new petechiae appeared.

That evening the patient presented a startling appearance. Intracutaneous extravasations too large to be covered by the outspread hand were present over the thorax and extremities, and extravasations of smaller size were present on the face. The patient could not be roused. The temperature was 99°F., the pulse 140, and the respirations 30. It was believed that the patient presented the classic clinical picture of the Waterhouse–Friderichsen syndrome.

During the night she gradually went into a state of vasomotor collapse, and in spite of further infusions of 5 per cent glucose in physiologic saline solution and 2 cc. of cortical extract, she died at 6.25 a.m. on the following day.

Permission for autopsy was not granted, so that doubt remains whether there were adrenal hemorrhages, as required to fulfill the criteria of the Waterhouse–Friderichsen syndrome. The duration of the illness was approximately 47 hours.

The diplococci seen in the blood smears were cultured from the blood stream and found to be meningococci.

DISCUSSION

Fulminating meningococcemia runs its course in a matter of hours, usually less than forty-eight. Of this form of meningococcal infection, Herrick[1] has

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said, "No other infection so quickly slays." In this disease the diagnosis must be made as promptly as possible and treatment instituted at once, for if the development of the clinical picture is awaited, the hope of survival is negligible. The length of time required for blood cultures reduces their value as a clinical aid[2]; as in this case, they are frequently of only academic and post-mortem interest.

McLean and Caffey[3] found meningococci in smears taken from the petechiae and purpuric extravasations seen in many cases of meningococcal infection. This procedure is simple and should be oftener resorted to. Unfortunately, many cases of meningococcal meningitis fail to develop a rash, and in fulminating meningococcemia the type of purpura is in itself almost diagnostic.

Herrick[4] and Adams[2] cursorily mention the finding of meningococci in the peripheral blood. Boone and Hall,[5] Marangoni and D'Agati,[6] and Kwedar[7] have encountered cases and presented photographs of meningococci in the peripheral blood. Thomas[8] mentions that in 6 of 12 fatal cases of fulminating meningococcemia organisms were seen in the peripheral blood. It is thus seen that a diagnosis from the blood smear is possible.

It is not suggested that every blood smear made in an acutely ill case should be scrutinized for phagocytized gram-negative organisms, but such examination is worth consideration when there is meningitis in the community, when there is objective muscle tenderness, and when a grippe-like or influenza-like syndrome is presented in combination with a leukocyte count that is predominantly polymorphonuclear. With reference to these points, it is impossible to be too alert in suspecting meningococcal infections when the disease is established in a community. The muscle tenderness that was present in this case was not sufficiently appreciated, but from observation of other cases of meningococcal septicemia it is apparent that Dickson et al.[9] are correct in their statement that this is a symptom that differentiates meningococcal septicemia from other forms of acute infection. The type of the tenderness is clearly different from the myalgia complained of in the usual case of acute febrile illness. The leukocytic response is likewise instructive, and Herrick[4] says, "Excepting lobar pneumonia no other acute infection is accompanied by such early and profound leukocytic reaction, 15,000 to 60,000 with an average of 90 per cent polymorphonuclears."

A bacteremia so overwhelming as to present organisms free in the blood stream in such numbers that they can be found on a direct blood smear offers little hope of recovery. The finding has been reported only in fatal cases.[5–8] As has been pointed out, however, if routine examination of the blood smear can establish the diagnosis, valuable time may be saved and therapy instituted in time to save the patient.[5, 6]

The suggestion that the examination of the direct blood smear in the occasional case may be worth while has added weight inasmuch as, according to Thomas,[8] "It seems fairly certain now that every clinical meningococcic infection with the possible exception of a variety of infection of the upper respiratory tract consists at some stage of an invasion of the blood stream by the causative organism." This concept is only now being generally accepted, but Herrick[1] clearly stated it in 1919. Indeed, the blood stream may be the only site of the infection. "Three patients," he states, "each having a blood culture positive for meningococci of Group I, recovered spontaneously without drug or serum therapy before the culture was known to be positive."

In speaking of fulminating meningococcemia, the subject of the Waterhouse–Friderichsen syndrome is called to mind. The terms are not synonymous. The Waterhouse–Friderichsen syndrome is an overwhelming septicemia that produces purpuric subcutaneous extravasations and bilateral adrenal hemorrhages. Although it is true that meningococcal infections usually produce this syndrome, overwhelming sepsis due to other organisms occasionally gives rise to similar adrenal hemorrhages. Furthermore, not all patients who die with the clinical picture of the Waterhouse–Friderichsen syndrome present adrenal hemorrhages. Thomas[8] remarks their absence in 6 of 23 autopsied cases. Hence it seems ambiguous to use the term "recovery" in speaking of the Waterhouse–Friderichsen syndrome, since the adrenal hemorrhages can only be inferred if the patient recovers. Clinically the term "fulminating meningococcemia" seems preferable to "Waterhouse–Friderichsen syndrome," and if the latter has any usefulness it should be restricted to pathological discussions.

The bilateral adrenal hemorrhages seen in these fulminating septicemias have suggested that the mechanism of death is that of acute adrenal failure. The hypotension, hypothermia or hyperthermia, vascular collapse, abdominal pain, vomiting and cadaveric cyanosis — incident to the intracutaneous extravasations — are indeed similar to the findings in the crisis of Addison's disease. There are, however, in the literature no significant data concerning electrolytes, blood sugar, nonprotein nitrogen and hematocrit readings in cases of fulminating septicemia. Aegerter[10] in reviewing the subject of the Waterhouse–Friderichsen syndrome calls attention to the paucity of such data. This is not to be wondered at, since the short course of the disease, the uncommonness of the cases in adults and the imperativeness of treatment leave little time for investigation. In the case reported herein the blood sugar was 89 mg. per 100 cc., and the nonprotein nitrogen 44 mg., the latter figure being somewhat elevated. It seems doubtful that changes in electrolyte or carbohydrate metabolism drastic

enough to produce death would occur in a matter of an hour or two. Especially doubtful does this explanation of death become when one considers that the experimental animal withstands complete surgical ablation of the adrenal glands for a much longer period than the forty-eight hours that is usually set as the upper limit of survival in fulminating meningococcemia.

On the other hand, the suggested treatment of these cases with massive doses of cortical extract, adrenalin, intravenous glucose and saline solutions and blood transfusions[10] seems worthy of trial. This type of therapy was used in the present case, but the amount of cortical extract was insignificant and the amounts of intravenous fluids were small. Loeb[11] states that 50 to 75 cc. of commercial cortical extract must be used every twenty-four hours in the treatment of crisis in Addison's disease, and a comparable dosage seems to be indicated in treating fulminating meningococcemia.

The hypothesis that death is due to acute adrenal failure is attractive but not proved. The additional fact that some patients die of fulminating meningococcemia without showing adrenal hemorrhages indicates that however undesirable the term "toxemia" is, there is still a place for this concept. In a bacteremia with organisms free in the blood stream in enormous numbers the products of their metabolism in terms of toxins are certainly appreciable, which suggests that antibacterial and antitoxic serums should be used in these cases. Thomas,[8] although admitting that the subject is controversial and that the majority opinion is indifferent to the use of antiserum, states, "There is one group of patients who seem to have been benefited by meningococcus antitoxin, namely, . . . [those with] septicemia."

It may yet be established that the subcutaneous extravasations seen in cases of fulminating meningococcemia are due to a vascular toxin. Thrombosis of small vessels in the adrenal glands by clumps of meningococci has been demonstrated, but proof that the skin lesions are due to a similar mechanism has not been forthcoming.

Summary and Conclusions

A case of fulminating meningococcemia has been presented.

The term "Waterhouse–Friderichsen syndrome" appears to be ambiguous when applied to the living patient.

The concept of acute adrenal failure as the mechanism producing death is attractive, although not proved, but certain measures of therapy useful in combating the crisis of Addison's disease may, nevertheless, be applicable in fulminating meningococcemia.

In this type of meningococcal infection antiserum should probably be used.

In certain cases the examination of the blood smear may establish the diagnosis more promptly than is possible by any other means.

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MEDICAL PROGRESS

THORACIC SURGERY (Concluded)

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THE ESOPHAGUS

ALTHOUGH the esophagus has been called "the last frontier of surgery," notable advances have been made in the surgical treatment of various lesions of this organ since the publication of the excellent and exhaustive review of the subject by Bird[48] that appeared in 1939. The achievements in congenital atresia and carcinoma have been outstanding.

Congenital Atresia

Vogt[49] classifies the anomaly of congenital atresia as follows: complete absence; atresia without tracheal fistulas; atresia with fistulas between the trachea and the esophagus, situated between the upper segment and the trachea, between the lower segment and the trachea or between both segments and the trachea. In this classification the second and particularly the third type constitute the great majority of reported cases that are being treated surgically with increasingly encouraging results.

The constant symptoms, consisting of regurgitation of milk and mucus with attacks of choking, cyanosis and coughing, occur when the infant is first fed. A plain roentgenogram of the abdomen shows the stomach filled with air, thus proving the presence of tracheoesophageal fistula, which occurs in about 75 per cent of the cases of atresia, as distinguished from the far less frequent complete or incomplete atresia of the esophagus without a fistula which occurs in 25 per cent.[48] As suggested by various authors,[50,51] diagnosis can be verified simply and safely by passing a small catheter into the esophagus. This proceeds readily into the stomach in any normal infant. If it meets obstruction at the level of the thoracic inlet, — this may be verified by fluoroscopy, — the diagnosis can be definitely made. With diagnosis thus established, attempts to visualize the blind pouch by injecting barium or iodized oil should not be made, since they almost always result in the introduction of opaque material into the lungs, with resultant pulmonary complications. Once the diagnosis is made, unless there are other anomalies that contraindicate operation, this should be proceeded with at the earliest feasible moment to prevent the frequent complication of aspiration pneumonia.

Although the ideal treatment is the ligation and division of the fistula with re-establishment of esophageal continuity, this cannot always be achieved, and other and indirect measures have been devised and successfully carried out to meet such situations. To Leven[52] must go the credit for the first successful extrapleural ligation of a tracheoesophageal fistula with cervical esophagostomy, which he performed in stages from January 5 to March 27, 1940, after a preliminary gastrostomy. At almost the same time, Ladd[50] independently worked out an essentially identical procedure that was equally successful. According to Humphreys[51] both these patients and several others operated on since that time by the same surgeons are still living at more than four years of age. With this plan of ligation of the fistula, esophagostomy and gastrostomy, an anterior thoracic esophagus is eventually constructed of skin by a series of plastic procedures, so that ultimately the patient conveys food and liquids from mouth to stomach by means of esophagodermatogastrostomy.

As stated above, the theoretical ideal is the establishment when possible of esophageal continuity by direct anastomosis. The first reported attempt was that made by Shaw[53] in 1939. Although the operation was successfully performed, a reaction from an elective transfusion resulted in death on the twelfth postoperative day. Haight and Towsley[54] report the first case successfully treated by extrapleural ligation of the tracheoesophageal fistula and end-to-end anastomosis of the esophagus.

In a recent personal communication, Dr. C. Haight states that he has been able to anastomose the two segments of the esophagus in 16 patients, 6 of whom are living; all of them have a patent esophagus. Five of these take all their feedings by mouth without difficulty, but in 1 case there is a high-grade stenosis for which retrograde dilatations are done from time to time. The sixth patient has a wide-open esophagus, but a recurrent tracheoesophageal fistula has been discovered. The oldest living patient was operated on three years ago and is doing extremely well. The youngest living patient has passed nine months since the operation.

Stricture

Simple severe stricture of the esophagus, the result of the ingestion of caustics or other severe cicatrizing trauma, has long been a discouraging problem. Frequent bouginage, a gastrostomy life or both are frequently the lot of these patients, many of whom are children or young adults otherwise in excellent health. Although the construction

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of an anterior thoracic esophagus from skin has frequently been accomplished with satisfactory functional results, the method has many disadvantages. Not the least of these is the appearance of fistulas at the site of anastomosis of the skin tube with the cervical esophagus and especially with the stomach. Many ingenious technics have been devised, but the constant regurgitation of acid gastric contents into the skin tube of the reconstructed esophagus sets up an irritation that sometimes results in ulceration of the tube and formation of a fistula at the anastomosis. These fistulas are difficult to heal and therefore usually impede satisfactory reconstruction of the esophagus.

Recently Yudin,[55] director of surgery of the Sklifasovski Institute for Traumatic Diseases in Moscow, has reported on the surgical construction of an artificial esophagus in 80 cases. He recommends drawing a segment of jejunum through an anterothoracic subcutaneous tunnel as high as the angle of the left inferior maxilla, where it is immediately or at a subsequent stage anastomosed with the cervical esophagus. This author states that in his opinion a total intestinal plastic operation is the one of choice and is practicable in strictures located below the middle of the cervical section of the esophagus. The construction of the new esophagus should be carried out in two stages: exteriorization of the intestine — through a skin tunnel — up to the neck and direct anastomosis with the cervical esophagus. He believes that the operation should not be completed in one stage because of the tremendous risk of infecting the whole subcutaneous tunnel at the point of anastomosis of the esophagus and the jejunum. By July, 1943, Yudin had created artificial esophaguses in 88 cases, total intestinal esophagoplasties being performed in 21. The direct operative mortality was 2.3 per cent.

Carcinoma

In 1940, Garlock[56] wrote, "Heretofore, the diagnosis of carcinoma of the esophagus was tantamount to signing the patient's death warrant, and the physician advised palliative measures consisting of gastrostomy, radiation therapy or both." Because of the pioneer work and enthusiasm of this surgeon and many others, such a situation no longer obtains, and many probable cures have been accomplished in a disease that was once held to be beyond help and hope. The successful surgical resection of a cancer-bearing portion of the esophagus, particularly the lower and midportions, has been so frequently reported in the past five years that it may now be accepted as having passed beyond the stigma of a surgical stunt. It has been estimated that 5 per cent of all malignant disease occurs in the esophagus, with the proximal third involved in 18 per cent of these cases. Experience with radiation therapy has given no encouragement, and surgery has afforded the only hope of a cure, so that the surgeon must do all in his power to establish an earlier diagnosis.

It has been customary to divide the esophagus into three parts, and to speak of the upper, middle and lower thirds. Churchill and Sweet[57] believe that it is more useful to divide it into fourths, the second and third fourths being thought of as the middle half. From the point of view of surgical management they consider three zones. Zone 1, the upper fourth, extends from the base of the neck to the superior surface of the aortic arch. Zone 2, the middle half, includes that portion extending from just above the aortic arch to just below the level of the inferior pulmonary veins. Zone 3 includes the lower fourth of the thoracic esophagus, the cardiac orifice of the stomach and the adjacent few centimeters of the stomach, as well as the fundus.

Carcinoma of upper esophagus (*Zone 1*). Bird[48] states that two factors appear to have contributed to the recent lack of interest in carcinoma of the upper third of the esophagus, as compared to the enthusiasm shown in the attack on growths in the middle and lower thirds. In the first place, the cases are not so frequent; secondly, the field of operation is ill defined, and the lesion most frequently encountered — Trotter's type of hypopharyngeal epithelioma occurring in young and middle-aged women— is prone to be neglected by both the laryngologist and the thoracic surgeon. Hudson[58] reports 2 cases of carcinoma of the proximal portion of the upper third of the esophagus that illustrate the value of complete removal of the lesion and the disappointment that may follow anything short of complete removal. The lesions are so intimately associated with the larynx or involve it in such a manner that both the esophagus and the larynx must be removed if a cure is to be expected.

The results of surgical excision in the upper esophagus have not on the whole been encouraging, but they are good enough to stimulate continued attempts at cure and perfection of technics. Radiation is only of palliative value.

Carcinoma of midesophagus (*Zone 2*). Approximately four fifths of the lesions in carcinoma of the esophagus occur in its thoracic and abdominal portions.[59] In general it may be said that the greater part of the thoracic esophagus must be removed if a carcinoma of the middle third is to be cured, and the Torek[60] operation or some modification of it has usually been employed. Up to 1939, the only certain cure of a carcinoma of the midesophagus was obtained in 1913 by Torek,[60] whose patient lived for twelve years after operation and died of pneumonia without recurrence, as shown at autopsy. There are undoubtedly others, since Garlock[61] in 1941 stated that one of his patients was alive almost five years after resection. This operation entails preliminary gastrostomy and cervical

esophagostomy at the time of the transthoracic resection of the carcinoma, with the connection of these two stomas contemplated at a later date to re-establish continuity, usually by an anterior thoracic esophagus of skin.

There are many unsettled technical problems involved that need not be discussed in this review.

Churchill and Sweet[57] report 9 cases, with 3 patients alive and well. There were only 2 postoperative deaths. Ochsner and DeBakey[59] report 2 cases, with postoperative deaths in both. Phemister[62] reports resection and cervical esophagostomy in 4 cases, with 3 survivals. Wu and Loucks[63] have operated on 4 patients with carcinoma of the midesophagus, with 2 survivals. Santy et al.[64] explored 6 patients and were able to resect the esophagus in 2, both of whom survived. Allison[65] describes 4 cases of esophageal carcinoma treated by the excision of the thoracic esophagus in 3 and of the stomach and abdominal esophagus in 1. All the patients survived the operation, but none lived longer than one year. Franklin[66] successfully removed the thoracic esophagus for cancer in 2 cases. One patient is well, and the other died thirteen months after operation. Carter[67] operated on 2 patients with carcinoma of the lower third by a modified Torek technic with 1 death in the hospital from meningitis and 1 recovery. Garlock[61] has done 13 Torek or modified Torek operations with 6 deaths. One patient is alive almost five years later and another thirteen months later.

In a recent communication Garlock[68] states that he believes that greater mobility of the stomach can be obtained by dividing the branches of the left gastric artery so that the stomach can be pulled higher into the left pleural cavity without tension. He has also brought the proximal end of the esophagus anterior to the arch of the aorta and performed esophagogastrostomy in cases in which the Torek operation would formerly have been necessary. We have repeated this procedure at the Massachusetts Memorial Hospitals and have been gratified at the ease with which esophagogastrostomy can be performed with the aid of these maneuvers. It seems likely that continuity between the stomach and the esophagus can be established within the thorax in a greater number of middle-third (Zone 2) cancers by means of this technic than has hitherto been thought possible.

Carcinoma of lower esophagus and upper end of stomach (Zone 3). Carcinomas arising in the fundus or cardia of the stomach and extending to involve the lower esophagus pose an almost identical problem as do tumors arising in the lower esophagus itself, and operations on both types of lesions will be considered together.

According to Bird,[48] in spite of the fact that Biondi (1895), Sauerbruch in his early work and Janeway and Green (1910) had worked out an apparently satisfactory endothoracic esophageal-gastric anastomosis on dogs and cadavers; no successes in human cases were recorded until Ohsawa's in 1933.

At the 1938 meeting of the American Association for Thoracic Surgery, Adams and Phemister[69] reported the first successful thoracic esophagogastrostomy for carcinoma performed outside of Japan. This was for squamous-cell carcinoma of the lower third of the esophagus. The resection followed careful preparation by experimental work on dogs, and the authors had profited by experiences derived from a previous unsuccessful case.[48]

Since that time, an increasing number of successful cases is being recorded in the literature. In 1940, Garlock[56] reported resection and esophagogastrostomy in 2 cases with 1 recovery. He also performed radical resection and intrathoracic esophagogastrostomy in 3 cases of carcinoma originating in the cardia, and stated that he was convinced that the only logical method of treating cancer in this situation was by the transthoracic transdiaphragmatic route. In the same year, Carter et al.[70] reported 2 successful cases of transpleural esophagogastrostomy for carcinoma of the esophagus and for carcinoma of the cardiac portion of the stomach. Ochsner and DeBakey[59] have reported 2 cases, with 1 survival. Phemister[62] states that resection and esophagogastrostomy were performed in 4 cases, in 2 of which carcinoma was primary in the esophagus and in 2 primary in the stomach. One patient in each group survived the operation. Wu and Loucks[63] have operated in this manner on 6 patients with carcinoma involving the stomach or esophagus or both, with 4 survivals. Garlock[61] performed transthoracic resection with esophagogastrostomy for adenocarcinoma of the cardia and squamous-cell tumors of the distal esophagus. There were 15 cases with 6 deaths, an operative mortality of 40 per cent. Churchill and Sweet[57] report 11 cases in which resection and esophagogastric anastomosis for carcinoma of the stomach or lower esophagus was performed. There was 1 postoperative death, a mortality of only 9 per cent. Two of the survivors died of recurrent disease, but all the remainder were well and free from symptoms for from three months to two and a half years after operation.

Garlock,[61] in summary, states that the general problem of the surgical treatment of cancer of the esophagus can, on the basis of his experience, be reduced to exact figures. The average patient with cancer of the middle third stands a 54 per cent chance of surviving the operation and a 30 per cent chance of living for over one year. With squamous-cell cancer of the distal third, the patient has a 70 per cent chance of surviving the operation and an almost 100 per cent one of living for over two years. In the group of adenocarcinoma of the cardia, the probability of operability is only 37

per cent; there is a 60 per cent chance of operative survival and an 80 per cent probability of living for over one year.

As may be noted from the foregoing reports, there has been a great advancement in the treatment of this disease by these methods. It is reasonable to expect even greater improvement in operability, mortality and survival with earlier diagnosis and greater experience in operative management.

The Thymus Gland

There is no organ concerning which physicians have so little exact knowledge as the thymus gland. There has recently been some evidence of a relation between abnormalities of this gland and myasthenia gravis.[71]

Stimulated by the work of Blalock et al.,[73] who in 1941 reported the effects of removing the thymus gland from 6 patients with myasthenia gravis, the first concerted attack on this problem, Sloan[72] examined 350 thymus glands. These included approximately 200 specimens removed during routine autopsies at the Johns Hopkins Hospital and 150 specimens removed from patients who died suddenly and were autopsied at the city morgue. Sloan's findings, in part, are as follows:

Examination of 10 thymus glands, none of which contained a tumor, removed at operation from patients with myasthenia gravis showed constant changes. In all there was some age involution and an increase in the number of lymphocytes present in the thymus. In 7 cases, abnormal numbers of lymphoid follicles with germinal centers were present in the medulla. There was no epithelial hyperplasia and no constant change in the character and number of Hassall's corpuscles. In 6 autopsied patients with myasthenia gravis who had not undergone operation there was no generalized lymphoid hyperplasia. The changes found in the thymus gland in myasthenia gravis were not part of such a generalized lymphoid hyperplasia.

Clagett and Root,[71] reporting on 10 cases in which thymectomy was performed, with 1 death, state that their results have been satisfactory enough to warrant further employment of this operation in cases of myasthenia gravis and, at times, even in cases in which no thymic enlargement is demonstrable by x-ray. They discuss the advantages of the three approaches that they have used — transcostal, posterolateral and sternal splitting.

Hardymon and Bradshaw[74] summarize the literature on the value of surgery in 132 cases of proved myasthenia gravis, including 3 of their own. Approximately 50 per cent have shown improvement. They believe that more cases must be operated on before conclusions can be drawn.

At the 1944 meeting of the American Association for Thoracic Surgery, Blalock[75] reported on the effects of thymectomy performed on 20 patients with severe myasthenia gravis. Only 2 of these patients had tumors of the thymus. Approximately 8 showed striking improvement, whereas in the others there was only slight if any improvement.

Obviously much work remains to be accomplished on this fascinating subject. If a relation between the thymus gland and myasthenia gravis exists, there appears to be some factor that is not as yet understood.

The Heart

In most cases of chronic heart disease, the physician is not able to effect a complete cure but is able to prolong life and to make the patient more comfortable. There are, however, some diseases of the heart or of the structures indirectly affecting it that respond brilliantly to surgical therapy. These conditions constitute only a small percentage of the total, but this does not minimize the importance of diagnosing and treating them (Blalock[76]). It is with the surgical types of heart disease in which cure can often be effected that this review will be concerned.

Patent Ductus Arteriosus

The first attempt at obliteration of a patent ductus arteriosus was reported by Strieder[77] in 1937. The patient was operated on at the suggestion of Dr. Ashton Graybiel[78] and was already ill with subacute bacterial endarteritis. Death occurred on the fourth postoperative day and was due to acute dilatation of the stomach. The first successful case of surgical ligation of a patent ductus arteriosus was reported by Gross and Hubbard[79] in 1939. Since that time many successful cases have been reported, so that the operation has been established as a sound and rational procedure.

Burwell[80] has tentatively suggested the indications for the performance of this operation. These indications, which are generally accepted by most writers on this subject, are as follows: the presence of important limitation or actual heart failure, the demonstration that the patent ductus arteriosus is interfering with normal growth, or the presence of subacute bacterial endarteritis; and the demonstration of uncomplicated patency in itself, because of the probable utility of the operation in preventing progressive overwork of the heart and in preventing bacterial endarteritis in a young person. The latter constitutes a strong suggestion that operation should be considered.

In 1940, Jones, Dolley and Bullock[81] reported the ligation of 13 cases, with 1 death. In 1942, Humphreys[82] reported 16 cases, with no operative deaths. There was 1 late death from complications arising from the presence of a postoperative aneurysm of the ductus. Six patients were operated on because of subacute infection of the blood stream. Three of these were apparently cured, 1 was still in the postoperative period, 1 had died, and 1 still had positive blood cultures.

Touroff,[83] reporting on a series of 10 cases of patent ductus arteriosus complicated by infection, states that cures occurred in 7. There were 2 deaths from accidental hemorrhage and 1 failure ascribed to vegetations on the aortic valve. Thus, among 8 operative survivals there were 7 recoveries and 1 failure.

There have been many other reports of small series and single successful cases.

It seems likely that the operative mortality will be less than 10 per cent in the hands of experienced and skillful operators. Improvements in technic will undoubtedly lower this rate.

Gross[84] has recently described a carefully worked-out technic whereby in 14 cases he has successfully completely divided the ductus arteriosus. This, of course, is the method of choice, since it eliminates the possibility of recurrences by recanalization when the ductus has been treated by single or even double ligation.

Coarctation of Aorta

In an intriguing paper, Blalock and Park[85] describe 43 animal experiments during the course of which they divided the aorta below the arch and, turning down the left subclavian artery, anastomosed it with the distal portion of the aorta. There were 10 survivals of several months or longer. The authors suggest the possibility of using the common carotid artery in man to bridge a coarctation of the aorta, but believe that it should be considered only in human cases in which the outlook is very grave, since many patients with coarctation of the aorta have a fairly long life expectancy.

Trauma

In recent years there have been many reports of successful suture of penetrating wounds of the heart.[86–96] One of the largest series is that of Bigger,[86] who operated on 17 patients at the Medical College of Virginia Hospital. Eleven recovered, and 6 died. Bigger also collected, by questionnaire, reports of 141 patients operated on by other surgeons and found that 71 recovered and 70 died, a recovery rate of approximately 50 per cent, which he believes more nearly approaches the actual recovery rate than do the figures collected from the literature. Elkin[87] reports 38 patients with heart wounds operated on by him or his resident staff. Of these, 22 recovered and none of them had any residual symptoms referable to the injury.

Blalock and Ravitch[96] discuss whether the total of successful end results would not be greater if a more conservative policy regarding immediate operation were adopted in cases in which there is no active bleeding through the chest wound or into the pleural cavity. To put the matter otherwise, may operation be delayed and aspiration of the pericardium be used in cases in which the symptoms are due to tamponade rather than to continued active bleeding? Even if one decides to pursue this policy, facilities for immediate operation should be available and tamponade should not be allowed to persist for over two hours. If blood accumulates after aspiration, operation is indicated. This coincides with the following directions, recently prepared for medical officers in the United States Army relative to the treatment of penetrating heart wounds with resulting tamponade: aspirate the blood from the costoxiphoid route, if possible; repeat the aspiration if there is a recurrence; and if there is another recurrence, perform a cardiorrhaphy through an extrapleural exposure. Cases falling into this category, which have been or could have been treated conservatively, have been reported by Bigger,[86] Elkin[87] and Strieder.[97] It seems more likely, under these circumstances, that a coronary artery or vein has been wounded and the myocardium incised for only a portion of its thickness rather than that a complete penetration of the myocardium has occurred.

I have been asked by medical officers in the Army course in thoracic surgery how one can be sure, on performing pericardicentesis, that the aspirating needle is in the blood-filled pericardium and not in one of the heart chambers. This question is not so naïve as it might appear at first glance, since I have seen blood aspirated directly from a heart of unsuspectedly large proportions with the operator under the impression that he was aspirating a hemopericardium, which actually was not present. As in any procedure, one gains facility only by experience, but if the needle is advanced slowly by the costoxiphoid route, a definite sensation is transmitted to the operator as the pericardium is perforated and at this point blood is obtained at once as the plunger is withdrawn. If the needle is cautiously advanced a short distance farther, the movements of the heart can be felt against the tip of the needle, which should then be withdrawn to the original depth at which blood was first obtained.

It seems, however, that not operating on a patient with a wound of the heart is an extremely hazardous course to follow. Nelson[98] believes that all cases of penetrating thoracic wounds in which it cannot be determined positively that the wound has not reached the heart should have exploration performed.

Olim and Hughes[88] and Zerbini[89] have each reported a case in which it was necessary to ligate the left anterior descending coronary vessels to repair stab wounds of the right ventricle. Both patients recovered, and follow-up indicated that they were unaffected by the procedure.

Nonpenetrating wounds of the heart resulting in contusion and rupture have been discussed by Elkin,[87] Bright and Beck,[99] Beck,[100] Leinoff[101] and Barber.[102] These authors agree that this type of injury is a frequent, though often overlooked, cause

of death. Compression of the heart between the sternum and the bodies of the vertebras, as when a driver is thrown against the steering wheel in an automobile collision, is a frequent mechanism resulting in cardiac contusion or rupture. Bright and Beck[99] analyzed 152 cases in which death was caused by rupture of the heart, as shown by autopsy. They conclude that an accurate differential diagnosis between cardiac contusion and coronary disease is almost impossible. On the other hand if a patient who has previously been in good health receives an injury and develops signs of circulatory embarrassment, he should be considered as having sustained cardiac injury. Treatment consists in absolute rest and sedation, mild laxatives to avoid straining and digitalis as necessary for irregularities. Rupture, if it is not an immediately fatal lesion at the time of injury, ordinarily occurs in the second week. Immediate operation is indicated, hence a thoracic surgeon should be readily available during this period. The authors suggest a pericardial graft at the suture line if the myocardium is infiltrated or extensively bruised or softened.

Crynes and Hunter[103] believe that the supposed rarity of traumatic rupture of the pericardium is not correct. They think that the scarcity of information concerning pericardial rupture is probably due to the fact that it is usually but one of multiple injuries terminating fatally and in itself may or may not contribute to the cause of death. They discuss a case in which was discovered an extensive pericardial rent, occurring in the absence of evidence of serious damage to the thoracic or abdominal viscera. The patient, a boy, lived for two and a half years after a crushing injury with but few symptoms attributable to the lesion present at autopsy.

Decker[104] raises the question whether foreign bodies in the heart and pericardium should be removed. He reviews 109 cases of foreign bodies in heart and pericardium reported since 1900, and states that foreign bodies should not be removed simply because of their presence. No emergency exists in those patients who survive the original trauma, unless one chooses to consider that free bodies in the pericardial cavity, sharp needlelike bodies and large objects need immediate or early surgery. Indications for removal must be based on prognosis and review of the condition of the patient and the degree and duration of the disability.

Turner[105] reports a case in which a bullet, with its base in the myocardium, protruded into the chamber of the left ventricle. When the heart was exposed at the time of the original injury, the bullet could not be seen or palpated and the attempt to remove it was reluctantly abandoned. The patient was well twenty-three years later.

Suppurative Pericarditis

Suppurative pericarditis is not a rare disease, but it must be suspected and sought in cases of generalized or thoracic infection if the diagnosis is to made in a high percentage of cases. Strieder and Sandusky[106] reviewed 28 cases collected from the literature from January 1, 1934, to January 1, 1940, and added 10 cases from the Boston City Hospital, which brought the total of the recorded cases for which pericardiostomy had been performed to 265. The mortality for cases so treated is approximately 50 per cent. Without operation the mortality approaches 100 per cent. With the advent of chemotherapy the mortality with operation should be further reduced. When the diagnosis is made, operation should be undertaken forthwith. There is no reason for delay as there is in early empyema thoracis. It is unfortunate that some physicians are under the impression that chemotherapy together with pericardicentesis suffices to effect a cure in a large percentage of cases. If chemotherapy is of value as an adjunct, its effectiveness should be measurably increased by the prompt establishment of early, open, adequate pericardiostomy.

In cases with generalized infection, even with pericardiostomy, the prognosis remains that of the pre-existing infection, although, by relief of the cardiac tamponade, the operation may be a life-saving measure. Adams and Polderman[107] performed pericardiostomy on 3 patients with pneumococcal pericarditis, 1 of whom recovered. Of the 20 cases reported by these authors, 12 were secondary to pneumonitis or pneumonia, 6 to osteomyelitis, 1 to cholecystitis, and 1 to mastoiditis. In 5 patients with staphylococcal pericarditis an abscess was found in the heart muscle. The electrocardiogram may be of diagnostic aid in such patients. Malec and Midelfart[108] successfully performed pericardiostomy in a case of purulent pericarditis complicating streptococcal empyema. They advocate early adequate drainage and state that it rarely results in postoperative adhesive pericarditis, an opinion that is shared by Shipley.[109] In an operative experience with 8 cases, with 4 recoveries, I have not observed adhesive pericarditis as a late complication.

For purulent pericarditis the low anterior approach with the resection of the fourth and fifth left costal cartilages is probably the best, and is the one oftenest used. Donaldson[110] has recently advocated a modification of the old Allingham approach for the drainage of nonpurulent effusions.

Constrictive Pericarditis

Since Churchill's[111] report in 1929 of the first successful resection of the pericardium for con-

strictive pericarditis in the United States, many other successes have been recorded, and the operation is now the accepted treatment in suitable cases of this disease. Blalock and Burwell[112] have published an important review of the subject, to which the reader is referred. In it they discuss 28 cases of constrictive pericarditis that they observed at the Vanderbilt Hospital in Nashville, Tennessee. The etiologic agent was established in 21 cases by aspiration, operation or autopsy, and was found to be the tubercle bacillus in 18 cases and *Staphylococcus aureus* in 3. Twenty patients had pericardectomy. Twelve were cured or greatly improved, whereas 8 died from several hours to two years after operation. These results are in accord with previous observations that approximately 60 per cent of the patients are either cured or greatly improved by the operation.

Harrington and Barnes[113] discuss the diagnosis and surgical treatment of chronic constrictive pericarditis and report 9 cases. Three patients were cured, 3 were improved, and 3 died, the deaths occurring three, four and twenty-nine days, respectively, after operation. Heuer and Stewart[114] have reported 7 operations without a death. Three of the patients were cured and 4 were markedly improved.

Tumors

Gebauer[115] reports a case of a huge intrapericardial teratoma containing a large, probably infected dermoid cyst that he removed at operation from a twelve-year-old girl. The patient died of uncontrolled hemorrhage from the aorta, which was inadvertently injured.

Beck[116] has successfully removed an intrapericardial teratoma. He also describes a case of a tumorlike mass in the wall of the left ventricle. The identity of the lesion was not established, but it was probably not a true neoplasm. This seems to be the first case in which a benign lesion of the heart has been recognized clinically and the only one in which removal of the lesion has been effected surgically. The patient was in good condition eighteen months after operation. Aneurysm of the left ventricle had not developed.

Angina Pectoris

Attempts to improve the blood supply to the ischemic heart by the establishment of a collateral circulation by surgical measures have continued. Unlike total thyroidectomy and alcohol injections about, or operations on, the sympathetic cord and rami of the upper thoracic region, which treat only the symptoms of angina, these attempts are based on physiologic principles and are directed at the underlying disease.

Beck[117] was the first surgeon to apply these principles. His original operation consisted in grafting the pedicled pectoralis major muscle to the heart. Schildt et al.,[118] experimenting on dogs to produce new communications between the coronary arteries by the application of inflammatory agents to the surface of the heart, found that powdered asbestos was the most satisfactory substance. It reduced the mortality and the size of the infarct following ligation of a coronary artery. Recently, Beck[119] has utilized this work and describes a new operation in which he abrades the parietal pericardium with special burrs and introduces powdered asbestos to further adhesions between the heart and mediastinal fat. He believes that fat is probably the best tissue for purposes of vascularization, but that all tissues that come in contact with the heart serve as grafts.

Feil[120] appraises the results in 37 patients with sclerosis of the coronary artery and angina pectoris operated on by the Beck technic. There were 14 postoperative deaths, a mortality rate of 38 per cent. Nine patients survived the operation but died four months to six years later. In 5 of these cases the results were excellent, and in 4 there was little or no improvement. Fourteen were still living, and of these, nine were definitely improved and 5 showed an increase in exercise tolerance and diminution in symptoms. The results in the 23 patients who survived the operation were as follows: excellent, 14 cases (61 per cent); good, 5 (22 per cent); little or no improvement, 4 (17 per cent).

O'Shaughnessy,[121] of London, whose brilliant work was terminated by his death in Flanders before Dunkirk while serving with the British Army, used the omentum as a vascularizing agent in an attempt to increase the circulation to the heart. He reported his results in 20 cases. Fifteen of these patients were suffering from angina pectoris; 5 of these died and 8 of the remaining 10 were free of angina, 7 having returned to work.

Strieder and Clute[122] have operated on two patients using the O'Shaughnessy technic. In one case there was an excellent result, but death occurred six months after operation as a result of an operation for diaphragmatic hernia, which was a complication of the original cardio-omentopexy. The second patient was still alive four years later, with only slight improvement in the angina but with a greatly increased exercise tolerance.

Thompson and Raisbeck[123] found that in animals adhesive pericarditis can be regularly produced by the introduction of sterile talc into the pericardial sac. Ligation of the descending ramus of the left coronary artery and vein at a point 1 cm. below the origin of this artery carried a mortality of 50 per cent. If this operation was repeated but in addition talc was introduced, the mortality was 25 per cent. In another group, talc was introduced and two or three weeks later a similar ligation of the artery was performed. In this group there were no deaths. Beck[119] repeated these experiments but was unable to obtain the same bene-

ficial results as these authors report. Thompson and Raisbeck have, however, reported gratifying results in a group of patients incapacitated by their angina, who were completely or partially relieved of pain and were able to return to work.

I have introduced aleuronat paste into the pericardium of a woman who was completely incapacitated by angina pectoris. She is alive and doing her own housework four years later. Another patient, a man suffering from angina pectoris secondary to syphilitic aortitis, has been treated by introducing talc into the pericardium. At the time of operation he was unable to walk the length of the ward without precipitating an attack, but one year later he was able to walk four miles without pain. He died in cardiac failure one year after operation, and autopsy showed complete obliteration of the pericardial space with a rich collateral vascular system.

A somewhat different approach to this problem has been used by Fauteux,[124] who found that if he ligated the magna cordis vein of the dog's heart and ligated the descending ramus of the left coronary artery, the mortality was much less than if he ligated only the artery. The important practical result of this procedure was a rise in arterial pressure in the entire apical region of the heart, the area drained by the surgically occluded vein. Fauteux and Palmer[125] ligated the great cardiac vein in 6 patients. One, a man of fifty-four, was working as a janitor two years after the intervention. Five others survived the operation for shorter periods of time. All were relieved of pain.

There is no implication that ligation of a vein will prevent thrombosis in an atheromatous but patent coronary artery, but there is evidence that venous ligation will cause changes in the vascular bed, such that, if thrombosis does subsequently occur, infarcts may be limited in size if not actually prevented from forming.

This highly important and controversial subject is still in a state of flux, and much work remains to be done. Many more patients must be operated on and the results must be carefully appraised before significant conclusions can be drawn. Beck[126] best sums up the matter as follows:

> Our interest should be focused on the point as to whether or not a reasonable quantity of blood can be made to pass from the grafts into the heart, regardless of the procedure and regardless of the tissue used. . . . We have been able to demonstrate vessels between the grafts and the heart, however, we have no way of knowing how much blood went through these channels. If these channels are numerous enough and large enough to transport a reasonable quantity of blood, then the principle upon which the operation is based would be established. If the principle were satisfactorily established, then the best method for doing the operation would become important.

Intracardiac Surgery

Harken[127] believes that recent advances in thoracic surgery suggest that it is time to take up experimental intracardiac surgery. Working in the surgical research laboratories of the Boston City Hospital, he has devised an ingenious method of producing bacterial endocarditis by attaching a metal clip to the posterior mitral leaflet. The clip is introduced through the left auricular appendage. Following the injury to the valve leaflet, the character of the endocarditis and the specific nature of the vegetations were directed by the administration of sulfanilamide derivatives and subsequent injection of the desired bacterial strains. Harken[128] has also devised a new cardioscope by means of which it is possible to visualize in the experimental animal the chambers of the heart, the valve leaflets, the clips in place and the chordae tendineae with moderate facility. It is entirely within the realm of speculation that some day cardioscopy will play an important part in surgery of the heart.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 30371

PRESENTATION OF CASE

A thirty-three-year-old bartender was admitted to the hospital because of jaundice.

The patient was apparently well until about six weeks prior to admission, at which time he noted that he tired easily and lacked his usual vigor. His appetite became poor, and he had episodes of morning nausea and vomiting. He also developed muscle cramps in the legs, low backache and brief cramplike epigastric pains just to the left of the midline. Two and a half weeks before entry he was told that the whites of his eyes were becoming yellow, and this was followed in a few days by a noticeable yellowing of the skin, which continued to increase. There was no associated pruritus, and there was no history of chills or fever. The urine became dark, but the stools remained normal in color. Frequent doses of magnesium citrate were taken for constipation. A few days prior to entry he noticed that the abdomen was enlarging and felt firm. On the day of admission a small amount of bright-red blood was noted in the stools.

For the preceding ten years the patient had been a bartender and was in the habit of drinking about a pint of liquor, supplemented with ten to twelve glasses of beer or ale, daily. He often went without food for several days at a time, although consuming the usual amount of alcohol. He smoked heavily — two or three packages of cigarettes daily — but denied taking drugs. There was no history of intolerance to fatty foods, abdominal distress or previous attacks of jaundice. The patient had noticed occasional swelling of the ankles in recent months.

The family history revealed that his mother had been operated on for gallstones and that his father, also a heavy drinker, had been confined at a state hospital for several years.

Physical examination on admission revealed a well-developed rather obese man who was intensely jaundiced. The skin was lemon yellow rather than orange or bronze. A few small spider angiomas were present on the anterior chest and right upper arm. The palms of the hands were erythematous, especially at the periphery. The tongue was beefy red and slightly smooth. The heart and lungs were clear. The abdomen was moderately distended. The liver edge was tender and palpable a handbreadth below the costal margin and also to the left of the midline. The knee and ankle jerks were absent, and there was hypoactivity of the deep tendon reflexes of the upper extremities. The calves were tender to pressure.

The temperature was 100.4°F., the pulse 88, and the respirations 24. The blood pressure was 100 systolic, 65 diastolic.

Examination of the blood revealed a red-cell count of 3,490,000, with 12.5 gm. of hemoglobin. The white-cell count was 11,700, with 85 per cent neutrophils. The urine had a specific gravity of 1.010 to 1.020, with negative to ++ tests for albumin and a ++++ test for bile. The sediment on one occasion revealed numerous bile-stained coarsely granular casts; no leucine or tyrosine crystals were seen. The urine urobilinogen varied from negative to positive in a 1:16 dilution. Although one clay-colored stool was seen on admission, subsequent specimens were brown, soft and formed, often with a positive guaiac test. The van den Bergh test was 8.0 mg. direct and 22.0 mg. indirect. The serum protein was 5.7 gm. per 100 cc., with an albumin-globulin ratio of 1.0. The cephalin-flocculation test was + in twenty-four and ++ in forty-eight hours. The alkaline phosphatase was 5.0 Bodansky units per 100 cc. The nonprotein nitrogen was 33 mg. per 100 cc., and the cholesterol 134 mg. The prothrombin time was 38 seconds (normal, 18 to 20 seconds). A blood Hinton test was negative.

Roentgenographic examination of the abdomen revealed no evidence of stones, but there was definite enlargement of the liver and spleen. The patient was treated with transfusions, dextrose and Amigen intravenously and vitamins and choline by mouth. He had one transfusion reaction. His temperature varied between 100 and 102°F., with morning remissions.

On the tenth hospital day the spleen was palpable, and in the following days the spleen became larger while the liver appeared to diminish in size and to become more tender. Definite ascites was demonstrable. The van den Bergh was now 12.4 mg. direct and 18.0 mg. indirect. The nonprotein nitrogen rose to 47 mg. per 100 cc., and the serum protein fell to 4.7 gm. The cephalin-flocculation test was + in twenty-four and ++ in forty-eight hours. The chloride was 86 milliequiv. per liter. The fasting blood sugar was 110 mg. per 100 cc. before and 117 mg. after the administration of 0.7 cc. of a 1:1000 adrenalin solution.

The patient continued to vomit and to complain of epigastric pain. Several stools contained gross blood, and on the sixteenth hospital day a clay-colored stool was noted. He became semicomatose and died on the nineteenth hospital day.

*On leave of absence.

Differential Diagnosis

Dr. Friedrich W. Klemperer: This is, in brief, the story of a thirty-three-year-old man who for ten years had lived on alcohol and inadequate food. Then, without obvious reason, he proceeded suddenly, within two months, from good health to a death due to dysfunction of the liver. Practically all the symptoms, physical signs and laboratory data can be explained by dysfunction of the liver and avitaminosis. He had a short prodromal period — three and a half weeks — of vague complaints. He then became increasingly jaundiced and constipated and the liver began to enlarge. We do not know whether the liver had been large before this episode. He had had swelling of the ankles for several months. This is hard to interpret. It may have been due to cirrhosis of the liver. It could, however, be adequately explained on the basis of avitaminosis and protein deficiency. We know that the serum protein was low. It may also have been on a cardiac basis, because a beri-beri heart is frequently seen in alcoholic addicts.

On admission he was intensely jaundiced and the skin was lemon yellow rather than orange or bronze. We know that the urine contained bile, and the lemon-yellow color is therefore explained by the combination of bilirubin jaundice and anemia. Spider angiomas are typical of liver disease. "The tongue was beefy red and slightly smooth." This represents evidence of niacin (nicotinic acid) deficiency. We also have evidence of thiamine deficiency, because he had absent knee and ankle jerks and hypoactive deep tendon reflexes of the upper extremities. The anemia may have been on a deficiency basis or may have been caused by blood loss from the intestinal tract. This bleeding could be adequately explained by the prolonged prothrombin time, again a sign of dysfunction of the liver. The renal function was good, as judged by the specific gravity of 1.020. Slight albuminuria was present, as often occurs in cases with marked damage to the liver. Bile was excreted in the urine, and also a small amount of urobilinogen, which indicates that bile passed into the intestine. The van den Bergh test gave high values, particularly by the indirect method, which is usually taken as an indication of damage to the liver cells and is evidence against an obstructive jaundice. We have further laboratory evidence of liver dysfunction in the low serum protein, particularly the low albumin, the positive cephalin-flocculation test, the prolonged prothrombin time and the decreased cholesterol. The adrenalin-glucose test simply means that there was not enough glycogen in the liver to be broken down to give a significant rise in blood sugar.

From the time of admission the course was gradually downhill, and the patient died the usual death of liver insufficiency.

The cause of this man's liver deficiency was most probably a combination of avitaminosis, protein-deficiency and alcoholism. A primary infectious disease, such as Weil's disease, can safely be ruled out because of the long afebrile prodromal period and the absence of nephritis. We are told that the patient had not taken any drugs, so that a toxic hepatitis due to something other than alcohol can probably be ruled out. There is one other possibility that I do not believe can be ruled out clinically and certainly not pathologically; that is, the hypothetical virus that causes so-called "catarrhal jaundice" may be superimposed on liver damage already done by the alcoholism and in this way cause a rapid downhill course. We have some clinical evidence against it — the relative length of the prodromal period and the absence of leukopenia and lymphocytosis. Because I know that the pathologist cannot tell this kind of jaundice from toxic hepatitis, I feel safe in ruling it out.

The question remains, Did the patient have cirrhosis of the liver or subacute yellow atrophy, that is, degeneration of the parenchyma of the liver? The course to my mind is somewhat too rapid for cirrhosis of the liver. The absence of evidence of collateral circulation does not help in differentiating the two conditions, because it hardly would have developed within two months. Ascites was not a predominating symptom, except toward the end of the disease. I cannot rule out a cirrhosis of the liver that was asymptomatic previous to the terminal episode, but there is no evidence in the record to support this view. I believe that, as occasionally is seen in alcoholism, the patient died of parenchymatous degeneration of the liver with fatty degeneration. Two months are enough time to give rise to regeneration and fibrotic changes in the liver, so that cirrhosis or beginning cirrhosis may also have been seen at autopsy.

Clinical Diagnoses

Subacute yellow atrophy of liver, with hepatic failure.
Alcoholic cirrhosis, early.
Splenomegaly.
Ascites.

Dr. Klemperer's Diagnosis

Toxic alcoholic hepatitis, with early cirrhosis.

Anatomical Diagnoses

Subacute alcoholic cirrhosis of liver.
Ascites.
Jaundice.
Hemorrhages into lung parenchyma.
Bile nephrosis.
Bronchopneumonia.
Cardiac dilatation.

Pathological Discussion

Dr. Benjamin Castleman: The autopsy on this patient showed, as was expected, a good deal of ascitic fluid. The spleen was quite enlarged, weighing 450 gm. The liver presented an unusual appearance. It was large, weighing 3500 gm., which is about two and a half times the normal size, and was olive green and moderately firm; scattered throughout the organ were several small, finely nodular areas, averaging 1 to 3 mm. in diameter. The large size of the liver, plus the fact that it was green, made one believe that this was an obstructive cirrhosis. It had all the gross characteristics of obstructive cirrhosis or a biliary cirrhosis, except that the bile ducts were perfectly normal in caliber and there was no evidence of extrahepatic biliary obstruction. At the time of autopsy we toyed with the idea that this might have been one of the rare cases of intrahepatic cholangitic biliary cirrhosis. Microscopic examination, however, showed that the gross observations were incorrect, and that the diagnosis was alcoholic cirrhosis. Almost every other cell contained the characteristic alcoholic hyalin that is seen in the degenerated liver cells, and there was a fair amount of fat scattered throughout. There was early connective-tissue formation, but not the dense fibrous tissue that one sees in long-standing cirrhosis. In other words this is what has been called a "subacute alcoholic cirrhosis,"* a liver on its way to the chronic stage. Perhaps a year before it weighed about 5000 gm., was full of fat and had little, if any, connective tissue; at the time of death it was beginning to shrink. There was a great deal of necrosis in the liver cells, with polymorphonuclear and lymphocytic infiltration, which one could argue was either an acute hepatitis or a toxic manifestation from whatever produces alcoholic cirrhosis. In any event acute degeneration was still going on.

There were many purpuric spots throughout the lung and slight focal bronchopneumonia. The kidneys were tremendous in size, each weighing almost 400 gm., and microscopically showed a bile nephrosis. The heart weighed 400 gm. and was markedly dilated, which possibly was a manifestation of vitamin deficiency.

CASE 30372

Presentation of Case

A sixty-nine-year-old Norwegian housewife was admitted to the hospital because of pain in the epigastrium.

As a result of the patient's poor memory the history obtained was rather inadequate. Fifteen years prior to admission she underwent an abdominal operation but was unable to state what had been done. Since that time pressure sensations and pain in the epigastrium had become increasingly frequent after meals, especially after the ingestion of greasy foods, fruit and desserts. Nine months prior to entry she noted increasing tiredness, associated with dyspnea on exertion and orthopnea. On consulting a physician, she was told that she had heart disease, and she was treated with digitalis and bed rest on and off for a period of three months, with apparent improvement. Three weeks before admission she developed severe epigastric pain after eating liver. The pain persisted and interfered with sleep, and she became increasingly weak, so that hospitalization was advised. She had lost about 20 pounds over a period of two or three years.

Physical examination on admission revealed an emaciated woman with a sallow complexion. Bilateral lens opacities were present. The pulse was flatly irregular, and cardiac murmurs were audible. Palpation high in the left upper quadrant revealed tenderness and a fullness that suggested a mass.

The blood pressure was 135 systolic, 90 diastolic. The temperature was 99°F., the pulse 90, and the respirations 20.

Examination of the blood revealed 13.8 gm. of hemoglobin and a white-cell count of 15,600, with 84 per cent neutrophils. The urine had a specific gravity of 1.020 and gave a + test for albumin; the sediment contained 4 red cells, 6 white cells and many epithelial cells per high-power field. The blood nonprotein nitrogen and sugar were normal. The serum protein was 6.1 gm. per 100 cc., and the chloride 94 milliequiv. per liter. A Hinton test was negative.

A gastrointestinal series revealed no intrinsic esophageal gastric or duodenal lesion. A mass was visible in the midabdomen, medial to the body of the stomach and tending to displace it to the left. No adhesions between the stomach and the mass could be demonstrated.

On the fourteenth hospital day an operation was performed.

Differential Diagnosis

Dr. Carroll C. Miller: The history and physical examination in this case are rather incomplete. We have no data that allow the pathology to be attributed to any one system of the body, except perhaps for two or three apparently minor details. The patient was an old woman who had apparently been going downhill for fifteen years, although the weight loss and indigestion were more marked during the two or three years prior to admission. It seems to me that we have to rule out certain conditions of the gastrointestinal and urinary tracts before going to the other systems and organs that might be involved. There was one x-ray study, a gastrointestinal series. May we see the films? I think that the description is clear enough in the record,

*Hall, E. M., and Morgan, W. A. Progressive alcoholic cirrhosis: clinical and pathological study of sixty-eight cases. *Arch. Path.* 27: 672-690, 1939.

but it will be interesting to see the exact location of the tumor mass.

DR. LAURENCE L. ROBBINS: I should be glad to talk about the tumor mass but I cannot see it; the statement in the record was probably based on an observation made during fluoroscopy. The body of the stomach is more to the left than usual, but I certainly cannot see anything that suggests a definite mass pressing against the stomach.

DR. MILLER: Was a pyelogram made?

DR. ROBBINS: No. I think that I can see the upper pole of the right kidney, which looks all right. This may be the left kidney. Since the films were not taken with that in mind, it is difficult to be sure about them.

DR. MILLER: Except for a possible connection with the stomach, an x-ray study such as this would probably be of significance only in localizing the tumor more accurately. Were any guaiac tests on the stools reported?

DR. BENJAMIN CASTLEMAN: No.

DR. MILLER: And no diastase or amylase tests?

DR. CASTLEMAN: No.

DR. MILLER: It seems to me that the discussion must come down in a rather vague way to two questions. What tumors occur in the left upper quadrant? What is the likeliest diagnosis in this particular case?

From the description of the x-ray films and the fluoroscopic examination, I suspect that the tumor was not in the splenic area. A kidney tumor, possibly a large cyst or a hypernephroma, may displace other viscera one way or another. The fact that there was no connection between the tumor and the stomach rules out fairly well a lesion in the stomach. The gall bladder is a curious organ in that it may, when enlarged, extend in a number of directions. Usually hydrops of the gall bladder extends downward and a mass may be found in the right lower quadrant, but it is unlikely to extend to the left in the region of the lesser curvature of the stomach. The retroperitoneal region must also be considered. The retroperitoneal tumors most generally found in this location are the fibrosarcomas and lymphomas. Then there are mesenteric cysts in the gastrohepatic and gastrocolic ligaments. Mesenteric cysts are most frequently found in young children and extremely rare in the sixty-nine-year-old range. The pancreas is the next likeliest, perhaps I should say the likeliest, location for a tumor that presents in the region of the lesser curvature of the stomach. Pancreatic cysts are fairly frequent and are usually not associated with any changes in blood chemistry, digestive processes or other physiologic reactions. They gradually increase in size, displace organs and finally become diagnosable either by palpation or because of pressure symptoms on adjacent organs.

Another diagnosis that is interesting to postulate is a tumor of the left lobe of the liver, such as a hepatoma. I have seen one such case in which the hepatoma presented by x-ray as a solid mass in the left upper quadrant medial to the stomach.

Are there any data in the history that lead us to a diagnosis of any one of these conditions? The first symptoms were pressure and pain. Naturally with a tumor of increasing size there might have been a sense of pressure, particularly after eating. As the stomach became filled with food there were symptoms of indigestion, particularly after greasy foods, fruits and desserts, which are more poorly digested than other foods. This could have been due simply to pressure against normal peristalsis. The suggestion of greasy food makes one think of a disorder of the gall bladder, liver or pancreas, but I do not believe that we have enough evidence of disease in either of these organs to be incriminating except perhaps for the pancreas, as I have said.

The pressure and pain were experienced intermittently and progressively over a fairly long period of time — three weeks before she was admitted. What is it in tumors and cysts that causes trouble? If the cyst has a pedicle it may become twisted and thereby infarcted. Its enlargement may cause pain owing to stretching of the capsule, and if it enlarges so as to press on its own mesentery it will cause various changes incident to gangrene or infarction. Infection is also a frequent complication of cysts. This again causes swelling and infarction if the process goes on long enough. The other eventuality is the development of malignant degeneration.

The laboratory data provide no significant information. The patient had a temperature of 99°F. — a bare elevation — and a white-cell count of 15,500, with 84 per cent neutrophils. This would go along with hemorrhage or with the early stages of infarction of a tumor. We do not know whether the urine was a catheter specimen. It gave a + test for albumin, and the sediment contained 4 red cells. If it was a catheter specimen, the presence of the red cells is important. A woman of this age may have senile vaginitis, with red cells in a voided specimen; if it was a catheter specimen, however, the red cells probably came from higher up. There is no other sign of involvement of the kidney, but the failure to take an intravenous pyelogram resulted in the missing of an opportunity to see a tumor. The blood sugar was normal. In pancreatic cysts there is often not enough damage to the pancreatic substance to produce a diabetic state or any change in the amylase or diastase level. The blood protein was normal, and the chloride slightly depressed.

My first diagnosis, a purely presumptive one, is pancreatic cyst. The next two possibilities, which are less likely, are a retroperitoneal solid tumor with malignant degeneration and a hepatoma.

DR. LELAND S. MCKITTRICK: I am not proud to carry on this discussion, and I am glad that Dr.

Miller was not much smarter than we were. When this woman came in she was miserable, as well as fibrillating, as it states in the record. We thought she had a carcinoma of the pancreas. I did not want to operate, but the family doctor, who was keener than we were, thought she ought to be operated on. I doubted that we could do more than establish a diagnosis.

Clinical Diagnosis

Carcinoma of pancreas.

Dr. Miller's Diagnosis

Pancreatic cyst?
Retroperitoneal malignant tumor?
Hepatoma?

Anatomical Diagnoses

Cholelithiasis.
Chronic cholecystitis.
Choledocholithiasis.
Chronic passive congestion of liver.
Anomalous lobe of liver?

Pathological Discussion

Dr. McKittrick: At operation the mass proved to be a congested liver. In retrospect, we paid too little attention to the fibrillation and to the possibility that it might have led to cardiac decompensation and an enlarged liver, thus accounting for the mass; in other words, we "missed the boat." But that was not all. What she really had was a gall bladder full of stones and a common duct full of stones, which are what the family doctor suspected. So through a left-sided incision we took out the gall bladder and removed the stones from the common duct. Then, thanks to the help of Dr. Conger Williams of the Cardiac Department, her heart failure was properly treated and she was not much the worse for wear. Of course we were extremely anxious to get her well after we had learned that she did not have cancer of the pancreas.

Dr. Castleman: The biopsy of the liver showed slight congestion, not severe failure, and I wonder if the mass was merely an anomalous lobe.

Dr. McKittrick: As a matter of fact, the patient had clinical heart failure, but some of us did not recognize it.

Dr. Castleman: The gall bladder showed marked inflammatory change.

Dr. McKittrick: This case provides one good lesson: one has to be very careful about getting the mind centered on a particular thing. Dr. Miller and I both centered our minds on the mass.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal

Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

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Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.04 per year (Boston funds); $8.52 per year for all foreign countries belonging to the Postal Union.

Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

ALKALINIZATION DURING SULFONAMIDE THERAPY

A number of investigators[1–5] have recommended the routine use of alkalies, in the form of sodium bicarbonate orally or sodium lactate parenterally, as an adjuvant to sulfonamide therapy. They have presented evidence to indicate that this tends to prevent crystalluria and its concomitant damage of the urinary tract. Daily doses of 15 to 24 gm. (average, 20 gm.) of sodium bicarbonate or 1500 to 2000 cc. of one-sixth molar sodium lactate solution (27 to 36 gr. of sodium lactate) are usually required to maintain the alkalinity of the urine for this purpose. It is claimed that clinical experience with these drugs has resulted in almost complete elimination of urinary-tract complications (crystalluria, hematuria, renal colic and anuria).

It is known, however, that the administration of such large doses of a sodium salt may, in certain types of patients, result in the retention of water in amounts that may be serious and even dangerous. This may be expected particularly in patients with cardiac, renal or hepatic insufficiency of even mild degree and often occurs in pregnant women. Indeed, Lyons and his associates[6] have shown that water retention may result in normal persons from the ingestion of similar amounts of sodium bicarbonate. These investigators gave 50 gm. of sodium bicarbonate to each of six normal subjects over a period of forty-eight hours. They found an average increase of 400 cc. in the plasma volume and an average increase of 1.6 kg. in body weight, an increase of 14.4 per cent in the former and one of 2.3 per cent in the latter. Slightly greater increases were found after the ingestion of 40 gm. of sodium chloride over a similar forty-eight-hour period. Edema due to water retention is also frequently observed postoperatively in patients receiving parenteral injections of sodium chloride solutions containing equivalent amounts of sodium. Furthermore, the recommended amounts of alkali have a tendency to increase excretion of the sulfonamide drugs, thus necessitating the use of larger doses of these drugs to maintain the desired blood concentration.[7] Should this increased excretion occur in a patient who also had water retention, the renal complications might be increased rather than eliminated, in spite of the greater solubility of the excreted drug in the alkaline urine.

Elsewhere in this issue of the *Journal*, Drs. Ohnysty and Wolfson present evidence that these difficulties may be overcome by substituting potassium salts, particularly potassium bicarbonate, for sodium salts in alkalinizing patients during sulfonamide therapy. The use of potassium bicarbonate has the advantage of producing the same alkalinity in the urine and at the same time having a diuretic effect,[8] instead of the water-retaining tendency of sodium salts.

The use of potassium salts instead of the sodium salts has been rejected by previous workers because of the possibility that abnormal cardiac

rhythm may result.[9,10] The observations of Winkler, Hoff and Smith,[11] however, indicate that the chief objection to the use of potassium salts arises from their possible irritating effect on the upper gastrointestinal tract. This irritation is probably of significance when potassium chloride or iodide is used, but probably is less important when potassium bicarbonate, acetate or citrate is employed. The findings of these workers indicate that serious cardiac complications or fatal poisoning from the oral administration of potassium is a distinct possibility only when there is complete, or almost complete, anuria associated with a high degree of nitrogen retention. They state, "The relatively slow absorption, the vomiting when large doses are given, the mode of distribution in the body and, most important of all, the continued ability of the most severely damaged nephritic kidney to excrete potassium all combine to make such poisoning very difficult to bring about."

The use of potassium bicarbonate, or that of the acetate or citrate if either is readily available, thus offers a useful adjunct to sulfonamide therapy and is certainly worthy of extensive clinical trial. Until more information concerning toxicity becomes available, it seems advisable to use small doses (5 to 10 gm. daily) and to administer these substances with care to nephritic patients who show evidence of moderate or marked nitrogen retention.

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QUALITY VERSUS QUANTITY PRODUCTION

The Maternity Center Association of New York, in the June issue of its bimonthly broadside *Briefs*, points out the advances that have been made in reducing maternal and infant mortality, as well as some of the dangers that continue to exist. Despite the difficulties imposed by war conditions, with shortages of physicians and nurses and overcrowding of hospitals, the maternal mortality in the United States reached a record low level of 2.1 per 1000 live births in 1943. Infant mortality also achieved a new low point.

This success is attributed to the following factors: the profit derived from studies of the causes of maternal mortality; public education; advances in obstetric knowledge, with new, important life-saving discoveries; the increasing rate of hospitalization; the Social Security Act, with the Government entering the field of maternity and infancy care on a grand scale; and the Emergency Maternity and Infancy Care Plan for the wives of men in the four lowest pay grades of the armed services.

Dangers that still block our arrival at a millennium are listed as follows: the continued existence of many poorly managed and poorly equipped small hospitals in both urban and rural areas; the continued high death rate of infants under one day of age, due largely to prematurity and birth injury; continued outbreaks of infectious diarrhea of the newborn; and lack of positive good health and hygienic living conditions for so many of our expectant mothers.

From the expansion of this theme—the reduction in maternal and infant mortality—the broadside goes on to a somewhat ambiguous discussion of the political aspects of birth rates. In this discussion the usual failure to distinguish properly between quality and quantity production is found. All political leaders, mindful mainly of muscular competition between nations, have viewed declining birth rates with apprehension and have rightly feared dwindling populations while their aggressive neighbors bred huge potential armies. The *folie raisonnante* of the blustering nations that needed more

babies to make bigger armies to fight for more territory for their expanding populations is typical of the type of inspired leadership with which we have no sympathy, although we must fear it. To enter into direct competition with it is serving no useful future purpose.

A declining population is no more undesirable than a rapidly increasing one—both lead eventually to race suicide, the one by stagnation or inundation and the other by mass starvation or total war, or both. A high fertility rate and large populations in themselves, moreover, are no guarantee against military exploitation by one's aggressive neighbors, as witness the plight of China and India.

The only reasonable answer is the more effective use of that intracranial organ by which man is supposed to be distinguished from the lower animals, for the control of population growths and selective breeding. When we use half the ingenuity in the breeding and feeding of man that we now direct toward the science of animal husbandry, we may find that brave new world in sight.

MASSACHUSETTS MEDICAL SOCIETY

The BLUE SHIELD

Medical societies in Kansas, Missouri and New Hampshire recently approved the establishment of nonprofit plans for the voluntary prepayment of medical and surgical bills resulting from hospitalized illnesses, injury and maternity care. This brings to eleven the number of states in which medical prepayment plans are co-ordinated with Blue Cross hospital-service plans. States in which medical plans were previously established and co-ordinated with Blue Cross plans are California, Colorado, Michigan, North Carolina, Delaware, Pennsylvania, Massachusetts and New York.

Of the above plans, by far the largest is Michigan Medical Service, sponsored by the Michigan State Medical Society. From recent reports one person in nine in Michigan is covered by this medical-care insurance, and during the four years of its life, Michigan Medical Service has paid to physicians in the state more than $6,000,000.

The Michigan plan is beginning to prove that the profession itself can solve the health problems facing the nation and that physicians operating under the present free system have something tangible to offer as a substitute for governmental medicine. Here in Massachusetts we have the same opportunity to solve the problem of a better distribution of medical care among the lower income groups and to forestall compulsory health programs but only if all the physicians in the Commonwealth give their enthusiastic and united support to the Blue Shield plan.

Doctors who have not yet become participating physicians are urged to communicate with the Blue Shield regarding participation in the plan. Doctors who have already become Blue Shield participating physicians are urgently requested to spread the gospel of Blue Shield protection among their patients and to distribute literature regarding the plan in their waiting rooms.

It seems unnecessary to add that the physicians who participate in the Blue Shield will be better off financially than they would be out of the plan. Figures are not available to show how much of the $6,000,000 of disbursements by the Michigan Medical Service would have been collected by the doctors themselves, but if it is true that only 80 per cent collections are made by the profession, then the Michigan physicians would have received only $4,800,000 for the amount of services.

The time to make an impressive record of achievement is now. When the war ends, and thousands of medical officers return to civilian ranks after witnessing the impressive and efficient distribution of governmental medicine to the 12,000,000 Americans in the armed forces, it may be too late. Furthermore, the 12,000,000 recipients of this same kind of medical care will undoubtedly have a good deal to say in the matter, but only if the Blue Shield has failed in its attempt to solve the problem.

In a recent survey in California, several interesting facts were established. Some of them are as follows: 88 per cent of the people think that the majority of doctors are doing a good job for the people; 34 per cent are against federal medicine; 50 per cent are in favor of federal medicine, and 16 per cent have not made up their minds. The question is, Why is there such a demand for federal medicine when there is such a high approval of the profession? The answer is, People believe that the cost of scientific medicine is too high. The conclusion is obvious. When something is needed by all the people but is too costly for them, eventually it is socialized so that it becomes available to all.

DEATH

JONES — Lombard C. Jones, M.D., of Falmouth, died August 17. He was in his seventy-ninth year.

Dr. Jones received his degree from Harvard Medical School in 1892. He was a member of the American Medical Association and the Harvard board of overseers.

His widow, a son, two step-daughters, two sisters and a brother survive.

(*Notices on page xix*)

NOTICES

THIRD POSTGRADUATE COURSE IN INDUSTRIAL MEDICINE

The Long Island College of Medicine, Brooklyn, announces the presentation of its Third Postgraduate Course in Industrial Medicine. This course will be given under the auspices of the Department of Preventive Medicine and Community Health during the three-week period October 16 to November 3, 1944, and will be conducted by more than fifty leading physicians in industrial practice, authorities in allied fields and members of the faculty of the college.

The main objective of the course is to provide physicians engaged in full-time or part-time industrial practice, as well as those who wish to enter this field, an opportunity to orientate themselves more fully to modern procedures in the rapidly developing specialty of industrial medicine. The two previous courses in this field, given by the college in the fall of 1942 and that of 1943, were presented as a wartime service to industry at a time when expanding production required increasing numbers of qualified physicians for industrial practice. The course this year will place particular emphasis on postwar conditions and problems associated with the return of workers from military service. Although designed for graduate physicians, the course will be open to industrial executives, personnel workers, industrial nurses, hygienists, engineers and to others interested in industrial health.

Enrollment in the course will be limited to sixty full-time students, and a tuition fee of $60 will be charged for the entire three-week course. A limited number of applicants particularly interested in specific phases of industrial medicine or with special qualifications will be accepted for admission on a part-time basis. All inquiries concerning the course should be addressed to Dr. Thomas D. Dublin, Department of Preventive Medicine and Community Health, 248 Baltic Street, Brooklyn 2.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, September 21

Saturday, September 23
*10:00–11:30 a.m. Medical staff rounds. Peter Bent Brigham Hospital.

Monday, September 25
*12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

Tuesday, September 26
*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

Wednesday, September 27
*12:00 m. Clinicopathological conference. Children's Hospital.

*Open to the medical profession.

October 2–5. Second Wartime Public Health Conference. Hotel Pennsylvania, New York City.

October 2–7. Seminar in Legal Medicine, Harvard Medical School. Page 110, issue of July 20.

October 3–5. American Public Health Association. Page ix, issue of March 30.

October 4. Medicolegal conference, Mallory Institute of Pathology, Boston. Page 110, issue of July 20.

October 9–20. 1944 Graduate Fortnight of the New York Academy of Medicine. Page xvii, issue of July 27.

October 10. New England Society of Anesthesiology. Page xvii, issue of September 7.

October 13. Mental Conditions Resulting from the War. Dr. C. A. Bonner. Pentucket Association of Physicians. 8:30 p.m., Haverhill.

October 16–November 3. Third Postgraduate Course in Industrial Medicine. Notice elsewhere on this page.

October 30. New York Institute of Clinical Oral Pathology, New York Academy of Medicine. Page 110, issue of July 20.

November 2–4. Association of Military Surgeons. Page xiii, issue of August 17.

District Medical Societies

SUFFOLK

December 7. Censors' meeting.

WORCESTER

October 11. Rutland State Hospital.
November 8. Grafton State Hospital.
December 13. Worcester City Hospital.
January 10. St. Vincent Hospital, Worcester.
February 14. Worcester State Hospital.
March 14. Worcester Memorial Hospital.
April 11. Hahnemann Hospital, Worcester.
May 9. Annual meeting.

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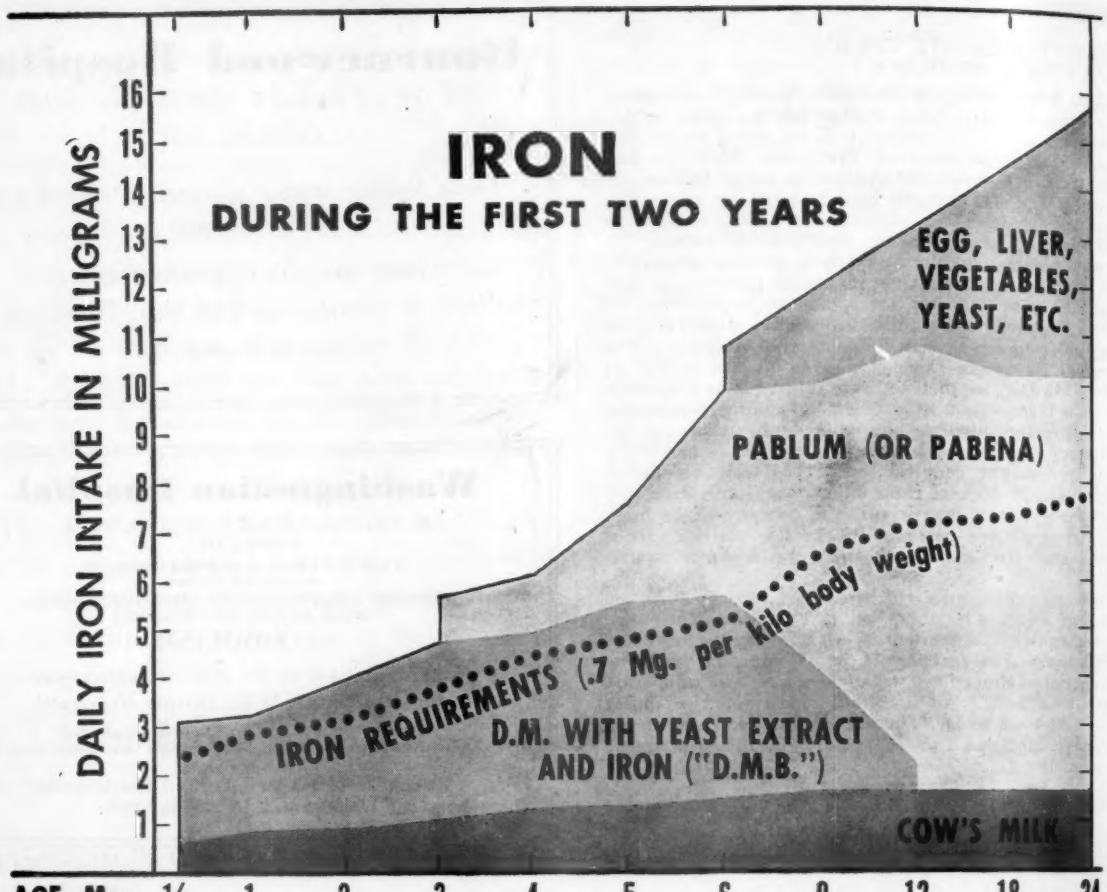

AGE, Mos.	¼	1	2	3	4	5	6	9	12	18	24
WEIGHT, Lbs.	7	9	10	12	14	15	16	19	22	23	25
MILK, Oz.	10	16	18	21	24	26	28	32	32	32	32
"D.M.B.," Oz.	1	1	1¼	1½	1½	1¾	1¾	1	¼	0	0
PABLUM, Oz.	0	0	0	⅛	¼	¼	½	¾	1	1	1

IRON DURING THE FIRST TWO YEARS

During fetal life iron accumulates (in the form of hemoglobin) in the infant's body. After birth the hemoglobin frequently drops to 50% by the third month, especially in prematures. Neither breast milk nor cow's milk is capable of offsetting this loss, as they are deficient in iron. This chart shows that when the carbohydrate and cereal supplements contain iron, a sizeable margin of safety over the requirements can be maintained, not only during the important first six months, but throughout the first two years of life.

More iron than the calculated requirement is needed because some iron is not utilized. In rapidly growing, or poorly nourished infants, and in the presence of infection, the need for iron may be even greater than is indicated in this chart for normal infants.

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